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NUTRITION AND THE EYE, EAR, NOSE AND THROAT.

(With Excerpts from the Literature.)

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This is the tenth editorial on this subject, and our annual comment has paralleled the general trend in Nutrition. At first vitamins monopolized the attention; we noted that their discovery sparked an awakened interest in foods and their direct bearing on health. In those days the literature on vitamins was so voluminous that it constituted a considerable portion of current medical literature. Of course, all nutrition studies are the concern of the physician regardless of his specialty, but much of this early literature dealt directly with the Eye, Ear, Nose and Throat.

Today the situation is very different — in contrast to only a decade ago. Now in order to uncover bits of news important to our specialties one has to search in strange and sundry places. They are certainly not to be found in Eye, Ear, Nose and Throat literature.

VITAMINS.

There is now a wealth of information about the vitamins present in food — but not of the vitamins created within the

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body by the intestinal bacteria. This is obviously of great importance in nutrition; also, anyone who prescribes the sulfa drugs or antibiotics is deeply concerned with their action on these bacteria. Although the behavior and meaning of intestinal flora are far from clear and the solution of this complex problem lies in the future, yet we can be guided by certain facts already available.

There seems to be no doubt that synthesis of vitamins has been proved in animals, and the general concept is that the same process occurs in man — although some consider that definite proof is lacking.

Najjar[1] expressed his opinion in 1945, after experiments on man as well as animals, and his further studies confirmed the former findings. His concept is given as follows: To a greater degree than generally realized vitamins are produced within the body. Production of vitamins occurs in the tissues themselves or within the bowel. Animals which do not require "C" in their food appear to synthesize it in their tissues. A simple example of countless observations is in the synthesis of "B_2" which is found in the milk in ten times the amount ingested by the cow. The synthesis of "B_2" also occurs in the feces of fowl, particularly after their passage from the bowel. When the sulfa drugs appeared it was found that they interfere with this intestinal synthesis of vitamins and it then became possible to study not only the separate vitamin requirements but also the extent of the synthesis. Merely suppressing such synthesis caused deficiencies that could not only be observed but also measured. Of great importance in man is the production of the B Complex by bacteria in the intestine. Man can synthesize "B_1", "B_2", nicotinic acid, biotin and folic acid. The ability to do this presents a new approach and a different outlook on man's food requirements. Obviously the amount of a vitamin in food is less than the physiologic need for it; the difference is accounted for by the amount synthesized. Clinically, the length of time sulfa drugs are ordinarily taken can hardly suffice to precipitate a state of vitamin deficiency; but the possibility of inhibiting synthesis long enough to have a deleterious effect should certainly not be ignored.

The experiments of Daft and Sebrell[2] indicate that "K" also is synthesized in the bowel. They comment that hemorrhage in the newborn, preventable by giving "K", occurs when the bacterial flora of the bowel has not yet become established; and that, later, intestinal synthesis of "K" ends this period of danger.

This problem, however, is far from simple. Recent experiments on the growth of chicks and poults[3-8] suggest that antibiotics actually help in the production of *more* vitamins — possibly by inhibiting some bacteria and aiding others. The poults grow faster and larger. A current concept is that certain bacteria utilize ingested vitamins; that these bacteria may be inhibited by the antibiotic; and so these vitamins remain fully available to the host.

ANTIBIOTICS.

It is only recently that two new side-effects have come to light. These are of direct importance to anyone who prescribes antibiotics. The time is not yet when it can be stated that antibiotics *1.* lessen the supply of vitamins and *2.* also permit yeasts and molds to flourish. However, among the limited number who have so far considered this matter, there is a trend of opinion in that direction.

Even in the very few years since the advent of antibiotics — it was only ten years ago that penicillin was introduced — the results have been not only good but startling. With this truly new epoch, first of the sulfa drugs and then of the antibiotics, the practice of medicine and surgery has been markedly changed. Occasionally certain unfavorable and even serious results have been noted, but from the standpoint of statistics and percentages the experience has been most favorable. By and large, the toxic reactions are not sufficiently serious to require that the antibiotic be stopped. The good far outweighs the bad. It is of course to be expected that any such powerful therapy would have some bad side-effects.

Deaths have occurred. To be sure, bad effects do not occur very often but they are caused by the antibiotic just the same.

Unfortunately, the patient who "just feels bad" confidently expects a "wonder drug" and a quick cure. This attitude has prevailed ever since the advent of penicillin — in fact even before that, when the sulfa drugs were introduced.

Our personal experience with bad side-effects has been mostly with streptomycin. We conferred with Dr. John S. Chase[9] on a large series of Veterans and it seemed to us that the streptomycin had attacked the Central Vestibular Tract,[10] as was the case in a physician whom we had studied. After taking streptomycin for a mild bladder infection he was seriously incapacitated for over a year and never fully recovered. This doctor, a neurologist, had the rare opportunity to study himself as a guinea pig. His hearing was normal. He had no vertigo. When he turned over in bed to his great surprise he kept on rolling. When he sat up in bed he kept on going and his head landed at the foot of the bed. "The brakes didn't work." When he gazed at an object across the room he could see it clearly but when he turned his head quickly the object jumped out of sight and he had to hunt for it. When he could attempt to walk he was unable to balance and reeled — in no particular direction. When he was able to ride in a wheel-chair he could not tell with the eyes closed when a corner was turned. The nurse rotated him in the wheel-chair but he had no sensation that he was turning. In brief, he had lost automatic ability to keep right side up, to maintain equilibrium, to recognize movement with eyes closed and to maintain fixation of gaze when the head was moved. After a year he prided himself on being able to put on his trousers standing in the middle of the room. By continual practice he has compensated considerably for his vestibular loss.

In spite of the remarkable benefits of streptomycin, especially in tuberculosis, it is apparent that like any antibiotic this rose has its thorn.

It already seems clear that after antibiotics are given monilia may flourish; and that they, normally present and inoffensive, seem to become pathogenic. Although there is as yet no explanation, the fact is that one may see oral lesions —

monilia by culture — promptly after giving an antibiotic. Also for some unknown reason, giving B Complex has a good effect — in both prevention and treatment. At the moment these might be termed "mysterious facts."

It seems strange that the concepts of a possible B Complex deficiency and the presence of lesions due to monilia have not been noted and reported in the past; it was less than two years ago that Harris[11] made the first comprehensive report. The possible deficiency and the definite lesions from monilia might well be termed "the two new Cautions" in the use of antibiotics. Our impression of this matter is that we should not make too much of it — or too little; but we should certainly be alert.

Although time may alter present concepts as new facts come to light, enough evidence is now available, as given in the following excerpts, to indicate that *1.* Physicians, especially otolaryngologists, should be watchful and aware of these two new "Cautions"; *2.* We should certainly be more hesitant in giving antibiotics so freely, especially for trivial ailments; and *3.* It would seem advisable to give the B Complex *with* the antibiotic especially when it is given over a long period. The B Complex itself can be given and, in addition, supplemented by foods that ars rich in the B Complex, such as liver.

If, as it now seems, antibiotics may be followed by moniliasis whereas sulfa drugs are not — one wonders what the reason might be. Antibiotics and monilia have something in common; antibiotics are derived from fungi — each one from a different fungus (with the exception of two which are derived from bacteria).

* * * *

Aureomycin was given orally by Harris[11] to 135 and chloromycetin to 46 patients with brucellosis. Many developed lesions in the mouth — aften within two to three days — first on the uvula, then the hard palate, pharynx, cheek, lip and tongue. The coating of the tongue changed from yellow to brown and eventually black unless treatment was interrupted.

Resolution of all oral lesions was of variable slowness; in some patients they persisted for several weeks. When the B Complex was given to a later group of patients such lesions occurred less frequently or resolved more rapidly. Later, when larger parenteral doses of the B Complex were given daily together with the antibiotics, the mucous membrane lesions occurred in fewer patients and in lesser degree. Harris ponders on the meaning of these lesions, set into motion by antibiotics. He considers that monilia may over-grow and gather virulence following the antibiotic effects on the intestinal bacteria; or that monilia may invade tissues whose resistance may have been lowered by a B Complex deficiency. He suggests careful supervision during, and for long periods after treatment with antibiotics.

It was Harris who first presented this new concept; he now writes us[12] that eighteen months subsequent experience confirms his original impressions, as follows: Big doses of B Complex, preferably parenteral, given with the antibiotic largely prevent monilial infection of mucous membranes as well as the manifestations of an apparent B Complex deficiency. This adds weight to the assumption that there primarily is a B Complex deficiency, with subsequent overgrowth of fungi. Further, when B Complex is given every other day lesions may appear, but if the dose is then given daily all lesions subside, even though the antibiotic is continued. He reasons that perhaps it is not only the vitamin deficiency which permits the fungi to flourish; possibly the elimination of normal intestinal bacteria plays a rôle, in that these may be natural antagonists of the fungi. Although purely an assumption, it may be that the fungi become virulent. Monilial lesions are not necessarily self-limiting even if the antibiotics are stopped. While they usually subside spontaneously, some require fungicidal treatment. One patient had steadily progressing mucous membrane lesions (monilia) for four months after the antibiotic was stopped. There was then a prompt response to fungicidal therapy. The rapidity with which mucosal lesions develop suggests to Harris that they could hardly be due to vitamin deficiency; and yet when B

Complex is given parenterally the lesions rapidly clear up. Also, the lesions exactly resemble those usually produced by a B Complex deficiency.

The same side-effects as these given by Harris were noted by Gewin and Friou;[13] in addition they observed prothrombin deficiency, visual disturbances with retinal changes and severe peripheral neuritis. Over a period of months they most carefully studied a patient in whom aureomycin and chloramphenicol effected a bacteriologic cure of endocarditis. After severe intestinal bleeding the prothrombin was 43 per cent; r.b.c, 2,100,000; hb, 7.0 gm. The eye findings included yellow vision, blurring and fading of letters while reading, spots in front of the eyes, slight blurring of both optic discs, granular changes in the retina and enlarged blind spots. These side-effects were attributed to deficiencies in the B Complex and "K". Vitamin therapy proved only partially effective until the antibiotics were stopped.

Increasing experiences with antibiotics are revealing a commensurate increase in toxic side-effects. It seems to Woods, Manning and Patterson[14] that one group of untoward effects is particularly deserving of emphasis and investigation. When bacteria are exposed to antibiotics, those susceptible are suppressed; but the non-susceptible may grow abundantly, even in the presence of high concentrations of the antibiotic. Much has been learned concerning bacteria but little concerning fungi; also there is scant information as to the effect of this suppression of bacteria on coexisting fungi. The authors encountered many cases of clinical moniliasis, in most of which the condition was apparently a direct sequel of antibiotic therapy. The first group of patients had sore mouth, hairy tongue, perleche — the clinical picture of thrush — resulting from the local use of penicillin or aureomycin as sprays, troches, soluble tablets or powders. These preparations have been widely prescribed by physicians and druggists and these reactions have been so frequent as to cause many physicians to discontinue their use. Monilia were obtained from the mouths of all. In a smaller group which developed diarrhea monilia were abundant in the stools. Of 25 patients the mouth

and throat were involved in 20. The sequence was sore throat, antibiotics (local or systemic), recovery; then after one to three days the oral and upper respiratory lesions already described. The tongue is most frequently involved and at first is thickly coated, often with an over-growth of the papillae, giving the appearance of a brown or black hairy tongue. Later the coating or hairy covering comes off and the tongue is left beefy red, dry and swollen with papillae standing out in relief. It is tender to palpation and on contact with acid juices and foods. The sense of taste is markedly altered. The buccal mucous membrane is at first covered with whitish exudate which can be wiped away easily. As this comes off it leaves the mucosa glazed and parched. Less often this exudate is seen on the tongue, soft palate, tonsils and pharynx. There are often small petechial hemorrhages in the mucosa. The palate, uvula, aryepiglottic folds and pryiform fossae are acutely red and edematous. In one case the exudate extended the entire length of the esophagus.

The chance of getting a deficiency on a good diet seems to Jukes[15] to be small, because we do not know of any way in which antibiotics would interfere with the assimilation of vitamins normally present in the food. The only change which might be produced by antibiotics would be to lessen production of vitamins by intestinal bacteria. Jukes considers that even this deficiency would not be noticed because of the vitamins provided by the food, in a preformed state. He further comments on the oral lesions occurring after a few days local use of antibiotics and considers that such deficiency could hardly be produced in so short a time; he regards such lesions as due to a local reaction to the antibiotic. As to monilia, he suggests that by eliminating bacteria an antibiotic may render available to yeasts and molds the nutrients otherwise appropriated by the bacteria.

Leitner[16] also states that it takes considerable time before definite deficiency symptoms and signs are produced experimentally in well-nourished healthy subjects. In spite of this recognized fact he reports a patient who, after aureomycin, showed gross angular stomatitis and cheilosis, painful tongue

— shiny, swollen and denuded. Large doses of the B Complex healed the cheilosis and stomatitis in three days; the tongue became painless though still swollen. It would be tempting to speculate, says Leitner, if such an acute deficiency is due to interference with biosynthesis — or to "anti-B" vitamins evoked by the antibiotic.

Impressed with the work of Harris, Tomaszewski[17] undertook a careful study of these two new side-effects of antibiotics. They occur most frequently following chloramphenicol and aureomycin. They are usually not of a serious nature. As the use of these two antibiotics is rapidly increasing, a study of the side-effects is important. There is a dramatic disappearance of bacteria, and the appearance of fungi — usually monilia. Observations were made on 126 cases. In each case bacteriologic tests first showed that the pathogenic organism was sensitive to both of these antibiotics. The tongue was scraped with a sterile slide every two or three days in all cases before, during and for one to three weeks after cessation of treatment. Cultures were made for identification of fungi and bacteria. Both antibiotics were given by mouth in capsules. The side-effects were seen more often in patients treated for a longer time and with large doses. In some cases diarrhea occurred shortly after the starting of treatment; more often the patients complained only of loose stools. In a large proportion of patients the stool became odorless after a few days and in some this continued for a time after the cessation of treatment. Several had lesions of the rectum, anus and vagina. The most frequent lesions, however, were oral. Dryness of the mouth was noted — similar to that produced by atropine; the amount of saliva was reduced. Changes of the tongue developed in the majority of cases. Usually after two or three days there was a gradual disappearance of the normal whitish coating and a denuding of the tongue. Later a fiery red, raw, beefy tongue with partial atrophy of the papillae was often seen. The lingual manifestations appeared more readily if the patient had been treated previously with penicillin or streptomycin. Soreness often started at the edges of the tongue. After stopping the anti-

biotic sometimes the tongue remained red and denuded for one, two or more weeks. In 10 per cent, some shortening of the papillae took place during the first few days' treatment and a brownish coloration appeared, gradually spreading over the surface. In four cases a true black hairy tongue developed. The sense of taste was often markedly affected. Redness of the mucous membrane of the mouth and throat often accompanied the changes in the tongue; also angular stomatitis. The changes in the mouth and on the lips were very similar to those seen in a deficiency of B Complex, especially riboflavin; so the vitamins were given and recovery seemed to proceed quickly, though not in a dramatic way. In some patients the B Complex was given together with the antibiotics; although the lesions were not prevented their intensity was definitely less. In most cases the bacteria disappeared in a few days after starting the antibiotic; at the same time fungi appeared in increasing numbers. In some cases monilia were found in large amount in the sputum, in the stool and in the urinary sediment. The normal bacterial flora usually return in one to two weeks after stopping the antibiotic, but fungi may persist for some days longer. The frequent coexistence of lesions in the three body orifices — mouth, anus and vagina — suggests some close relationship between them. The lesions may result from a chemical action on the local cells. At any rate absence of bacteria seems to permit the fungi to flourish.

In reporting a death from monilia Gausewitz, Jones and Worley[18] note that the patient had prolonged treatment with penicillin and then with chloromycetin. Autopsy showed multiple involvement of viscera by the monilia, by both histologic and bacteriologic study. They comment on the recent reports suggesting that antibiotics may act to increase the pathogenicity of monilia. Often monilia are isolated from healthy persons; they have been cultured from the lungs in nine of 71 routine autopsies. Stomatitis (thrush) is the most widely recognized manifestation; frequently the respiratory tract, vagina and skin are involved. Infections of various organs including the esophagus, bronchi and heart have caused death.

Fatal meningitis has been reported in four cases, in three of which monilia were identified by culture. In a meningitis patient who recovered monilia were seen in direct smears and identified in cultures of the spinal fluid.

* * * *

INTRAVENOUS FEEDING.

In our specialties intravenous feeding is rarely indicated but the internist and general surgeon have made advances in this field from which we can learn much. A book by Elman[19] concerns this subject only. At the turn of the century such procedures were limited to salt or glucose solutions. With advances in knowledge of nutrition parenteral feeding was devised for those who cannot eat, so that now all the six nutrients can be given by vein — water, salts (electrolytes), carbohydrates, fats, vitamins and proteins. Carbohydrate can be injected only when reduced to its basic form, glucose. Fats have been suitably emulsified although they are not yet available for general use; but mixtures of necessary amino acids are now available to furnish complete protein nourishment. Elman points out that a low level of "C' in the plasma does not necessarily signify clinical scurvy; this does result with the loss of "C" in the leukocytes. Determination of "C" in the plasma is relatively simple; but in the leukocyte it is more difficult and less standardized. This detection of "C" loss in the plasma permits treatment of scurvy in its "latent" phase, thus forestalling the onset of "active" scurvy. The need of "C" increases greatly after operation or injury. Large amounts of "C" are extravasated into the injured areas; it seems that the healing process requires much more "C" than is normally present in the tissues. This depletes the available "C". In extensive injuries such as burns, as much as 2 grams per day may be required to maintain a normal blood level, even though the total amount of "C" in the body probably does not exceed 5 grams. "B_1" is depleted by intravenous glucose and, like "B_1", riboflavin and niacin are also used up in

carbohydrate metabolism. Other vitamins are sometimes given; there is no objection since no deleterious effects occur. Some have gone so far as to inject crude extract of liver because it may contain unknown factors important in restoring the patient to normal condition. Although "A" is fat-soluble it is available in water-soluble form as carotene. There is a simple chemical test for "A". After severe hemorrhage plenty of fluid remains in the body; 11 liters are in the interstitial spaces and much of this is mobilized quickly. Unfortunately this fluid contains very little protein, so that the addition of this fluid to the circulating blood results in both anemia and hypoproteinemia. If a soldier dies after a sudden loss of 2 liters of blood one explanation might be that death is due to lack of 60 grams of plasma protein — the amount lost. Then the inability of the body quickly to replace this would be a tragic example of "too little and too late". Not only are amino acids the basic elements of all protein but they circulate in the blood in which they maintain a constant concentration just as glucose does. Amino acids are to protein what glucose is to starch. Glucose is the actual fuel and amino acids are the building stones which replace the wear and tear of tissue.

If the day comes when glaucoma and edema of the internal ear are conquered, it will be through knowledge and control of osmosis. The electrolytes (*i.e.* salts) are distributed in three spaces in the body, as mentioned by Jones[20] — the cell space, the interstitial space and the vascular space. Body water makes up about 70 per cent of the body weight; of this 50 per cent is in the cell space, 15 per cent in the interstitial space and only 5 per cent in the vascular space. We see at once that only the smallest space, the vascular, is accessible to us for measurement and treatment. What is going on in the other larger spaces, although our prime interest — as in glaucoma or hydrops of the internal ear — is something we can judge only indirectly. Present teaching is that sodium salts are found in the interstitial and vascular spaces while potassium salts are found in the cell space. Water moves freely back and forth between the three spaces in response to

changes in osmotic and circulatory pressures. The most important substances exerting osmotic pressure are sodium salts in the interstitial space, proteins in the vascular space and potassium salts in the cell space.

* * * *

"A".

There is little that is new about the well-known vitamins present in food. Of course water-soluble vitamins are freely eliminated whereas oil-soluble "A" and "D" are not. It has been known for years that overdosage of "D" can cause serious damage and many warnings have been issued. However, Caffey[21] now reports poisoning from excess of "A" — tender swellings, X-ray changes in the skeleton and increase of "A" in the blood. His patients received the usual concentrates of "A" and "D"; however, it seems that the "D" was not the toxic factor — because it has been used clinically in much higher doses without bad effects. Recovery from "A" poisoning is dramatically rapid — just from stopping the vitamin. Three types of concentrate commonly used were found to be toxic — if given in too large dosage. The hazards of such poisoning are considerably greater than the hazards of an "A" deficiency. Overdoing is worse than doing nothing.

A recent experience of Sulzberger and Lazar[22] concerns an adult. She had actually been taking 600,000 units of "A" daily for eighteen months. This self-medication was inspired by a radio commentator who recommended "A" as helpful in "keeping us from catching colds". Menses were shortened by three days. There were generalized joint and bone pains. Skin changes were the main feature — pigmentation, pruritus, dry rough skin with desquamation and excessive loss of hair including complete loss of eyebrows and lashes. The "A" was stopped at once; in addition, foods rich in "A" were restricted. Although vowing to cooperate she was reluctant to believe that anything as beneficial as a vitamin could actually cause trouble. She was convinced, however, when the joint and bone

pains, the pruritus and desquamation disappeared in ten days. Three weeks later hair began to reappear in all affected areas. After four months the patient appeared normal except for residual pigmentation. The physician who gives large doses of "A" should be on the lookout for signs of toxicity. If a patient is taking large doses for more than a few weeks regular blood examinations should be made for the "A" level.

* * * *

DEEP FREEZE.

Heat causes a loss of vitamins but such loss occurs much more slowly at low temperatures. Freshly frozen fruits and vegetables are far more beneficial than those that are processed or canned. There seems to be almost no end to the variety of frozen foods now available — fruits, vegetables, meats, poultry and fish. Lee[23] reports on their nutritonal value. Produce should be harvested in prime condition and should be stored at 0° Fahrenheit. It has been found recently that very quick freezing is not necessary; good quality and retention of vitamins result if the freezing point is reached in twelve hours. Home-freezing cabinets, therefore, meet the requirements.

* * * *

THE PATIENT.

The physician has opportunities to tell the patient, in as simple a way as possible, the message of food — good and bad. Such "popular" language is usually well received — whether acted on or not. In habits of eating, as in other habits, it is emotion and not reason that is apt to guide; however, any intelligent patient, concerned about his condition, is eager to learn facts. Many well-considered ideas are appearing in a series of radio broadcasts by the American Academy of Applied Nutrition.[24] In these talks nothing controversial is included but only the tenets and teachings of the Academy.

The public response shows that there is a surprising interest in Nutrition. Certain of these ideas are presented here as being helpful to a patient.

In vacation time the average child selects foods of poor value and eats irregularly; too often by the end of the Summer his condition is worse rather than improved. There is more food value in a dollar's worth of milk and eggs than in a dollar's worth of pie and cake. The child should have natural foods, not processed foods; and particularly, not refined sugar or refined flour.

The patient often asks: "What's wrong with sugar?" A safe answer is: "Refinement and Overindulgence". Of course even refined sugar has a definite value; it is well and accurately known as a source of immediate energy; however it is not a nutrient; it cannot be converted into body tissue; it is just fuel.

In their natural state there are many good sugars such as the raw sugars — cane, beet, maple, honey, date and fruit. "White sugar" today, however, means a refined product from which vitamins and minerals have been removed. As such it is no longer a nutrient. It provides calories only.

As to over-indulgence, the use of "sweets" has increased until now they provide one-seventh of America's calories — as sugar or in soft drinks, candy, ice-cream and bakery products. They satisfy the appetite and supplant valuable and needed nutrients. Our ancestors had no refined sugar; in fact they ate long before food processing of any kind had become a profitable business. The use of these unnatural foods of civilization has led to malnutrition and denegerative disease.

It is hard for most people to look upon refined sugar as a serious threat; few have even given thought to the matter or realized the extent of its use. Education of the layman is not the only need; many physicians, including pediatricians, provide candy suckers for their little patients and naturally parents and children come to consider this as approval of sugar.

Of the four tastes — sweet, sour, salt and bitter — by far the most liked is sweet. If one feels he must have sweets, saccharin is available, now so purified that it has no bitter aftertaste. It is 500 times sweeter than sugar. As it is not a food its use has no effect except to provide sweetness.

To "see the dentist" is not the answer to the tooth-decay which is present in 95 per cent of the population. The dentist accomplishes most when he guides the patient in the selection of good foods. It is believed today that the chief cause of caries is eating refined carbohydrates. The gums and sockets also go bad on refined carbohydrates and the teeth loosen and fall out. With rare exceptions, when bone is lost around a tooth it is not replaced.

Sunshine acts on the oil in the skin; this results in the formation of "D". If this oil is all "soaped away" this will not occur. The United States occupies only 6 per cent of the earth's surface — but has 92 per cent of the world's bathtubs. So it is quite possible that we overdo the bathing.

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THE INNERVATION OF THE COCHLEA (GUINEA PIG).*

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INTRODUCTION.

Since the time of Retzius, extensive work has been done on the innervation of the cochlea. On the basis of the course taken by the fibres, two types of peripheral processes emerging from the bipolar cells in the ganglion of Corti are described by von Ebner[1] and by Lorente de Nó[2]: *a.* the radial fibres which go directly to the hair cells and *b.* the spiral fibres with extensive ramifications.

a. The radial fibres (see 1, in Fig. 1) run directly from the ganglion cells to the inner hair cells where each fibre innervates several neighboring cells and each cell may be innervated by several fibres. Lorente de Nó states that these fibres carry impulses of a very definite "local sign." According to the observations of Held,[3] the innervation of the inner hair cells is richer and more complicated than that of the outer hair cells. Many fibres meet at this level forming such compact innervation that the course of individual fibres is lost.

b. The spiral fibres are grouped in four bundles: *1.* intraganglionic spiral bundle; *2.* internal spiral bundle; *3.* tunnel spiral bundle; *4.* the external spiral bundle.

1. The intraganglionic spiral bundle (see G.B., in Fig. 1) runs apicalward inside the ganglion of Corti for distances, established by several authors, as much as one-quarter of a

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turn or more. Collaterals are constantly given off to the organ of Corti, and according to Lorente de Nó the collaterals innervate, almost exclusively, the inner hair cells (see 2, in Fig. 1). The work of Rasmussen[4,5] shows that the so-called superior

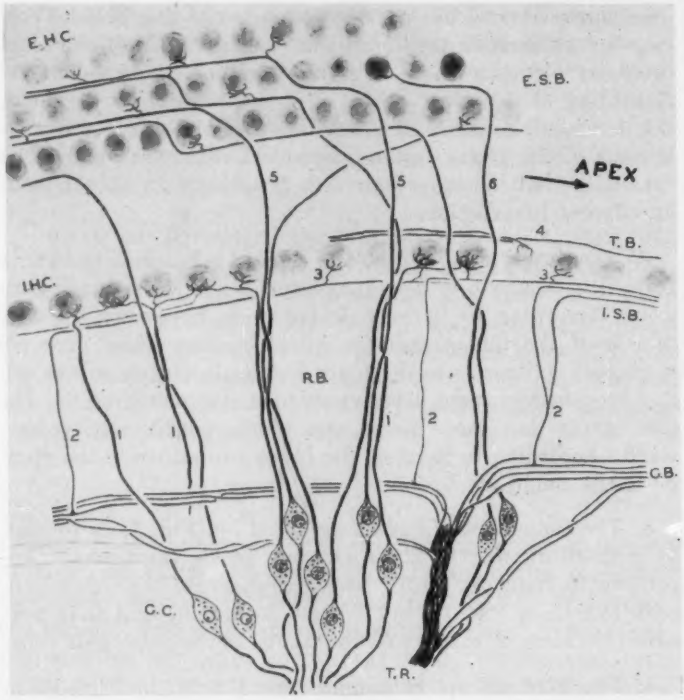

Fig. 1. The diagram represents the innervation of the cochlea based on observations of Lorente de Nó, Held and Rasmussen. E.H.C., outer hair cells; I.H.C., inner hair cells; G.C., ganglion of Corti; T.R., tract of Rasmussen which breaks up into several fasciculi which bend toward the apex and the round window to form the intraganglionic spiral bundle, G.B.; R.B., radial bundles formed by different types of fibres in their course inside the lamina spiralis. (1) Radial fibres to the inner hair cells. (2) Collaterals of the intraganglionic spiral bundle that later form the internal spiral bundle (I.S.B.) underneath the inner hair cells. (3) Collaterals from the internal spiral bundle to the inner hair cells. (4) Tunnel spiral bundle (T.B.), whose fibres turn back to innervate the inner hair cells. (5) Fibres to the outer hair cells that later form the external spiral bundle (E.S.B.) with multiple ramifications. (6) Fibre to the external hair cell, unbranched.

olivary peduncle has its origin in the superior[5] olivary complex and its terminals in the cochlea of the opposite side. The tract reaches the cochlea at the level of the first turn (see T.R., in Fig. 1) and enters the ganglion of Corti where it bends apicalward. He states also that morphologically the tract is formed by *efferent* fibres, a point of anatomical description which had been missed by all the previous workers in this field. From his observations[6] he presumed that the terminals of the tract could be identified with the intraganglionic spiral bundle. According to the observations of Lorente de Nó and others, the inner hair cells are innervated by radial fibres and by collaterals of the intraganglionic spiral bundle. It may then be concluded that the inner hair cells have both an afferent and an efferent innervation.

2. The internal spiral bundle (see I.S.B., in Fig. 1) is formed by a group of fibres coursing beneath the inner hair cells. According to Lorente de Nó these fibres are the terminals of the intraganglionic spiral bundle. They give off numerous collaterals to the inner hair cells (see 3, in Fig. 1) and occasionally some fibres cross to the outer hair cells. He also states that these fibres run almost exclusively apicalward. According to Retzius, the fibres run either to the apex or to the round window.

3. The tunnel spiral bundle (see T.B., in Fig. 1) is formed by a small number of fibres running inside the tunnel. According to Held, they turn back to innervate the inner hair cells (see 4, in Fig. 1), but Wever[7] points out that it is possible that some of them cross the tunnel to the outer hair cells.

4. The external spiral bundle (see E.S.B., in Fig. 1) is formed by fibres coming directly from the habenula perforata. The fibres cross the tunnel of Corti and reach the outer hair cells at the level of the inferior pole of the cells. The fibres then turn at right angles basalward, forming the external spiral bundle. These fibres have been thoroughly described by many authors; they are systematized as one bundle for each row of sensory cells. The fibres change their course constantly passing from one bundle to the next. The length

of individual fibres is as much as one-third of a turn. Held gives the most accurate description of their manner of innervation. According to his observations, there are two kinds of fibres: branched (see 5, in Fig. 1) and unbranched (see 6, in Fig. 1). The latter innervates one sensory cell while the former innervates several cells. The branched innervation predominates by far over the unbranched. In other words, there is a finely localized and a largely diffuse innervation of the outer hair cells. On the basis of innervation, he described two kinds of outer hair cells, those innervated by a single fibre and those receiving multiple innervation. There is not a systematic distribution of these two types, but they are intermingled with each other.

The original purpose of the present work was to establish as accurately as possible the size, length and systematization of the radial and spiral fibres and to incorporate the new concept of innervation determined by the discovery of Rasmussen. Later, during the histopathological studies of cochleas of animals subjected to acoustic trauma, our interest turned also to the effect of this injury upon the efferent fibres or terminals of the tract of Rasmussen.

METHOD.

The studies were carried out in young guinea pigs (seven to 30 days old). After perfusion of the animal with formalin, the cochleas were carefully dissected from the temporal bones. The pyridine and silver nitrate method of Cajal or the silver impregnation method of Bodian was used. With the technique of Cajal, the specimens were stained in block. Following dehydration and clearing, the cochleas were dissected under a binocular dissecting microscope (20×). The bone of the apical turn was first removed until the organ of Corti and surrounding structures were freed from the bony capsule; then the apical turn was removed from the modiolus. With fine, sharp instruments, Reissner's membrane, spiral ligament, limbus and modiolus were taken away. When the cochlea was properly dissected, the complete organ of Corti with the basi-

lar membrane retained its normal spiral shape. In order to avoid superimpositions, each spiral turn was divided into two or three pieces and mounted in balsam. The dissection was continued in the same way, turn after turn, until all of the cochlear duct was mounted on slides for study. When the method of Bodian was used, the specimens were fixed and stained in block; the cochleas were dissected as before and each piece was toned separately.

With training and with appropriate instruments, it is possible to dissect long pieces of the organ of Corti from the basilar membrane and surrounding structures. We used dissection because with this method the cochlea is represented as an uncoiled ribbon and it is easier to follow and measure the individual nerve fibres and to study their distribution at different levels. In serial sections of embedded material this is accomplished only with difficulty, because it is not always possible to trace the course of individual fibres.

The effects of degenerative processes of the cochlea on the intraganglionic spiral bundle were observed in stained and mounted sections of animals' cochleas used in studies on acoustic trauma. The animals had been briefly exposed at very high intensities and then kept alive for periods between three and four months. The material had been stained by the hematoxylin-eosin method. Although this method is not the proper one for the study of nerve fibres, it gives valuable information about the shape and number of cell bodies and fibres as a whole in the ganglion of Corti. The study showed that promising results could be expected by the use of silver stains in cochleas traumatized acoustically or subjected to other experimental methods.

RESULTS.

(A) The Radial Fibres.

In the preparations, it was possible to follow some of these fibres from the ganglion of Corti to the inner hair cells (see 1, in Fig. 2), where they innervate several neighboring sensory

cells. This is in agreement with the classical descriptions. The innervation of the inner hair cells is so compact that, as Held pointed out, it is not possible with the method used to follow accurately all of the individual fibres. For this purpose, it is necessary to resort to other methods of staining. It is clear, however, that the plexus formed by the nerve endings around the inner hair cells is remarkably more pronounced than on the outer hair cells.

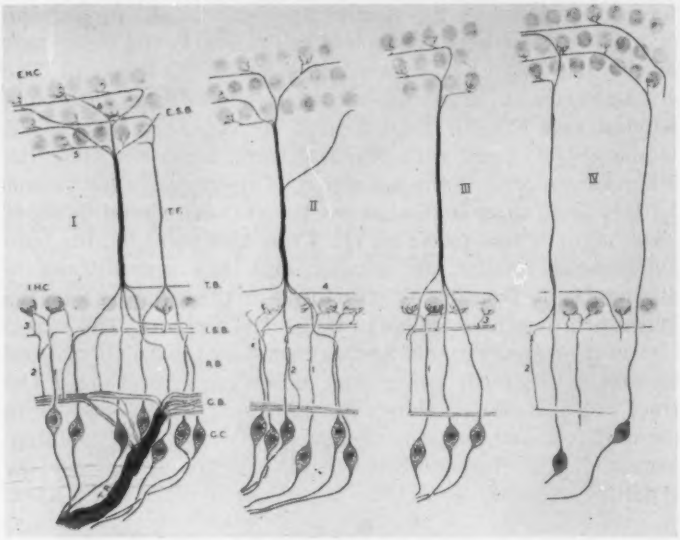

Fig. 2. The diagram represents the innervation of the cochlea of the guinea pig at different turns. It is based on our own observations and on those of Lorente de Nó, Held and Rasmussen. I, first turn; II, second turn; III, third turn; IV, apical turn. This figure is substantially the same as Fig. 1, but it emphasizes the fewer fibres of the intraganglionic spiral bundle above the first turn, the internal spiral bundle and the formation of fasciculi in the tunnel. Abbreviations are the same as in Fig. 1.

(B) The Spiral Fibres.

1. The intraganglionic spiral bundle. The terminals of the tract of Rasmussen have a spiral course within the ganglion of Corti anatomically identified as the intraganglionic spiral

bundle. The size and position of this bundle was determined in the adult guinea pig in both silver impregnated and hematoxylin-eosin stained material. The results were then compared with the animals subjected to acoustic trauma. The serial sections showed that the tract of Rasmussen enters the ganglion of Corti at about 3 mm. from the round window and breaks up into several fasciculi of different sizes and at different positions. After a short radial course some of them bend to the round window, others to the apex. Later the different fasciculi join together to form one bundle or a group of bundles. The bundle is found constantly in the outer part of the ganglion as is shown in Figs. 3, 5, 6, 7 and 8. The cross-sectional area of the bundle at 4 mm. from the round window (see Fig. 3) is 0.012 mm^2. At 7 mm. (see Fig. 5) it is about 0.005 mm^2. with borders clearly separated from the other structures. At the basal part of the second turn (about 12 mm. from the round window) the area is reduced to about 0.003 mm^2. or less (see Fig. 7). From this point on, the bundle becomes smaller and smaller, and it is very difficult to distinguish it from other structures in the ganglion; in the apical turn it is only visible in tangential sections. The reduction in the cross-sectional area is caused by the collaterals and terminals constantly being sent to the organ of Corti. The tract of Rasmussen is shown in Fig. 2 entering the cochlea in the first turn and breaking up into fasciculi to form the intraganglionic spiral bundle (G.B.). The figure also shows the greatly diminished size of the bundle as it reaches the apex.

In guinea pigs subjected to acoustic trauma and kept alive for long periods of time, atrophy of the organ of Corti and the primary neurons was found. The extent and location of damage varied with the frequency and intensity of the sound used, but the intraganglionic spiral bundle remained approximately normal in shape and size (see Figs. 4, 6 and 8). Measurements made in a few specimens showed a slight reduction in the sizes of the bundle in the basal part of the first turn. Unfortunately, there was no experimental material available which showed atrophy of the primary neurons to the end of the second, third, or apical turns.

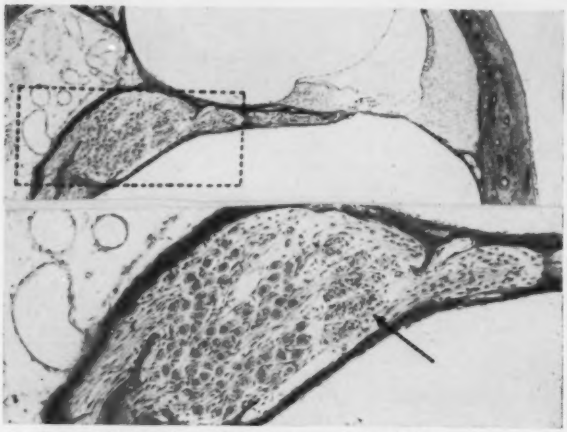

Fig. 3. Upper: Photomicrograph of normal basal turn at 4 mm. from the round window. Hematoxylin-eosin. 48×.

Lower: Photomicrograph of the area of the ganglion of Corti enclosed in the rectangle of the upper figure. The arrow points to the intraganglionic spiral bundle formed by several groups of fibres with their borders clearly outlined. 130×.

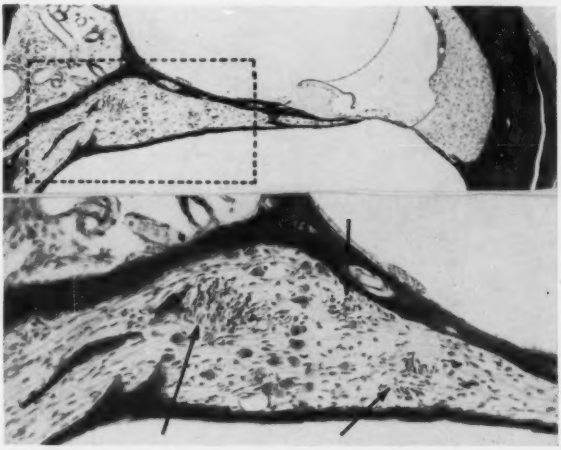

Fig. 4. Upper: Photomicrograph of pathological basal turn about 3 mm. from the round window showing atrophy of the ganglion and organ of Corti. Hematoxylin-eosin. 55×.

Lower: Photomicrograph of the area of the ganglion of Corti enclosed in the rectangle of the upper figure. The cells of Schwann remain and also a few ganglion cells. The arrows point to several fasciculi, situated at different places, with normal density of fibres. This illustrates the position of the fasciculi immediately after the tract of Rasmussen enters the ganglion. Later they join together to form a single bundle. 130×.

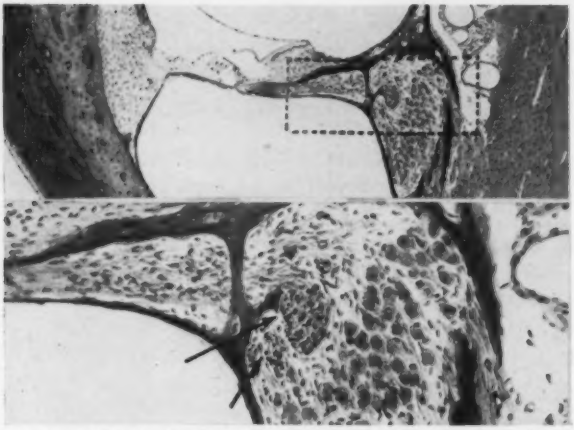

Fig. 5. Upper: Photomicrograph of the normal basal turn at about 7 mm. from the round window. Hematoxylin-eosin. 58×. Same animal as Fig. 3.

Lower: The arrow points to the intraganglionic spiral bundle formed by a big and compact fasciculus and two small ones. The total cross-sectional area of the bundle at this level is reduced to one-half of the size seen at 4 mm. from the round window. 180×.

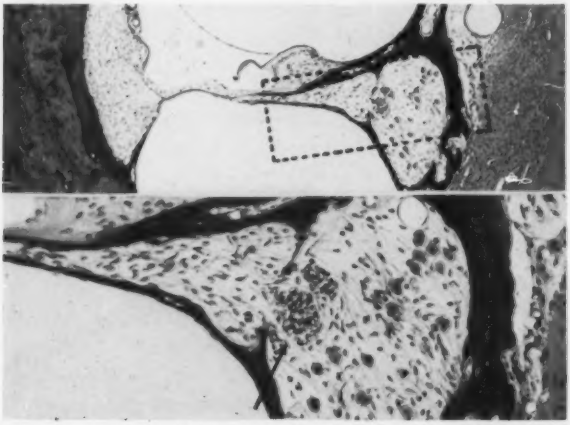

Fig. 6. Upper: Photomicrograph of basal turn, traumatized acoustically, at about 8 mm. from the round window. It shows atrophy of the ganglion and organ of Corti. Same animal as Fig. 4. Hematoxylin-eosin. 66×.

Lower: Higher magnification shows the cells of Schwann and a few ganglion cells. Arrow points to the intraganglionic spiral bundle formed by three fasciculi clearly outlined. The density of fibres is about the same as in the normal. 170×.

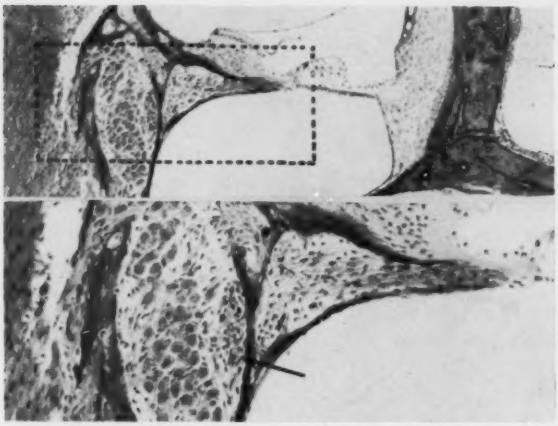

Fig. 7. Upper: Photomicrograph of normal ganglion and organ of Corti of the second turn at about 13 mm. from the round window. Same animal as Figs. 3 and 5. Hematoxylin-eosin. 65×.

Lower: The arrow points to the intraganglionic spiral bundle which at this point is already reduced to one-fourth of the size of the bundle at 4 mm. from the round window.

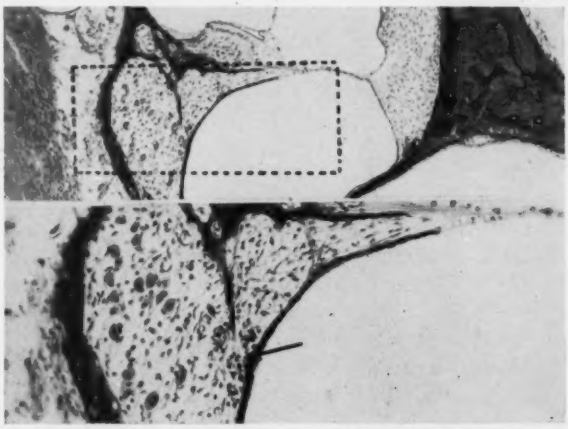

Fig. 8. Upper: Photomicrograph of pathological second turn at about 13 mm. from the round window. Same animal as Figs. 4 and 6. Teh organ of Corti is absent and the ganglion of Corti presents a pronounced degree of atrophy. Hematoxylin-eosin. 50×.

Lower: Schwann's cells remain and also a few ganglion cells. The arrow points to the intraganglionic spiral bundle which is reduced to a few fasciculi of small size but with a total cross-sectional area of about the same size as the normal represented in Fig. 7, lower. 166×.

The same pattern of atrophy of the organ of Corti and primary neurons was observed in slides in the collection of Schuknecht. His animals (cats) had received traumatic blows to the head. The intraganglionic spiral bundle remained approximately normal. Guild[3] described the persistence of the intraganglionic spiral bundle in humans after atrophy of the organ of Corti and primary neurons.

2. The internal spiral bundle. According to Lorente de Nó and others, this bundle runs apicalward underneath the inner hair cells. He also described it as terminals of the intraganglionic spiral bundle and stated that they innervate, almost exclusively, the inner hair cells. Our observations agree with these (see 3, in Fig. 2) although it was found that many fibres course basalward and many other fibres extend for distances of as much as 200μ or more in the internal spiral bundle. They then pass laterally to the outer hair cells. Lorente de Nó found the internal spiral bundle to be formed of very fine fibres. There is, however, a great variability in their diameters. Indeed, the finer ones (less than 0.5μ) are the most common, but there are also many with a diameter as great as the largest unmyelinated fibre (approximately 1.0μ) found in the cochlea.

3. The tunnel spiral bundle. This is formed by a small number of fibres coursing inside the tunnel of Corti. According to Held, they turn back to innervate the inner hair cells. It was found that the bundle runs in close relation to the inner pillars and is formed by a few fibres which are of small diameter (0.5μ). They extend either towards the round window or towards the apex (see 4, in Fig. 2). Some of them course for a distance of 300μ. All of them turn outwards to the outer hair cells. This fact confirms the possibility pointed out by Wever. In some specimens there was an increase in thickness of the bundle as the apex was approached. Fig. 11 illustrates the position and relative size of the bundle in relation to the internal spiral bundle in the apical turn.

4. The external spiral bundle. The present studies are in agreement with the observations of Held concerning the

course of these fibres and their innervation of the outer hair cells. There are two types of fibres, branched and unbranched, and also two kinds of cells, the singly and the multiply innervated. In only a few instances were unbranched fibres and singly innervated cells observed. The origin of the external spiral fibres is from different sources: *a.* directly from the habenula perforata; *b.* some fibres, after crossing the habenula perforata, bend either to the round window or apex for variable distances. These form in their course part of the internal spiral bundle before they turn outward to the outer hair cells; *c.* some collaterals, although exceptional, pass from the internal spiral bundle to the outer hair cells, and *d.* all fibres of the tunnel spiral bundle bend at right angles to the outer hair cells.

The course of the fibres in the tunnel is in two different positions; one crosses in fasciculi horizontally about the middle of the tunnel (medial fibres); the other crosses in single units or groups of two fibres at the bottom of the tunnel [basilar fibres (see Fig. 9)].

In the first and second turns, the fasciculi are formed by five to six fibres each (see Figs. 2, 9 and 10), but single units and fasciculi of two or three fibres are also intermingled with them (see Fig. 12). The fibres of the fasciculi come from different places as has been already described and as illustrated in Figs. 2 and 12. As soon as they cross the inner pillars, they usually join into a very tight and twisted fasciculus. When the fasciculus reaches the outer hair cells it breaks into single units which form the external spiral bundle. Most of the single units run basalward but many were also found to course in the opposite direction as illustrated in Fig. 13. The number of fasciculi which traverse the tunnel and the number of fibres in each fasciculus diminish as the apical turn is reached. In this turn, usually neither fasciculi nor single units are found to traverse the middle of the tunnel.

There is no information concerning the number of fibres to the outer hair cells at different levels. The present techniques do not stain all of the fibres.

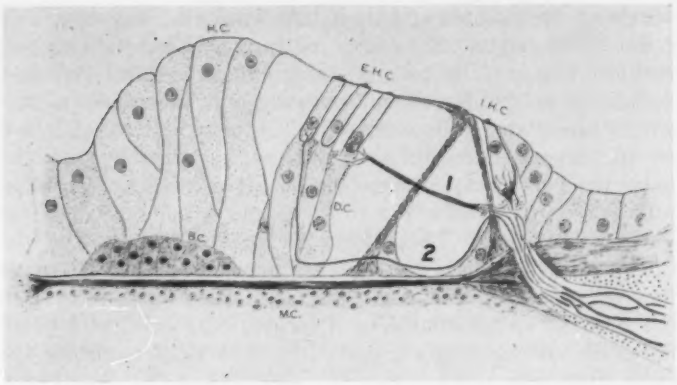

Fig. 9. Diagram of the organ of Corti in cross-section. It shows the structures and innervation as seen in the basal half of the first turn of the young guinea pig. I.H.C., inner hair cells; E.H.C., outer hair cells; H.C., Hensen's cells; D.C., Deiter's cells; B.C., Böettcher's cells; M.C., mesothelial cells. (1) Medial fibres crossing the tunnel in one fasciculus of four fibres, two coming from the habenula perforata, one from the internal spiral bundle (represented by dots underneath the inner hair cells), and the fourth one from the tunnel spiral bundle (represented by dots inside the tunnel close to the inner pillar). (2) Basilar fibre coming from the habenula perforata; in the tunnel it runs over the basilar membrane and then crosses to Deiter's cells, where it bends at right angles away from the basilar membrane toward the outer hair cells. The dots underneath the outer hair cells represent fibres of the external spiral bundle.

The basilar fibres (see 2, in Fig. 9) are fine fibres (less than 0.5μ) which pass between the inner pillars at the same level as the medial fibres. They extend across the tunnel immediately above the basilar membrane in single units or groups of two. They pass out of the tunnel at the foot of the outer pillars (see Fig. 14). Some course between Deiter's cells, bend at right angles and ascend to the outer hair cells, others bend when Deiter's cells are reached. They are present throughout the organ of Corti but their density increases as the apex is reached. At the apical turn almost all fibres to the outer hair cells are of the basilar type. After reaching the outer hair cells, the course of the fibres follows the same principle already described for the medial fibres. The exact information as to their endings or their origin behind the habenula perforata is not available. The particular arrangement of

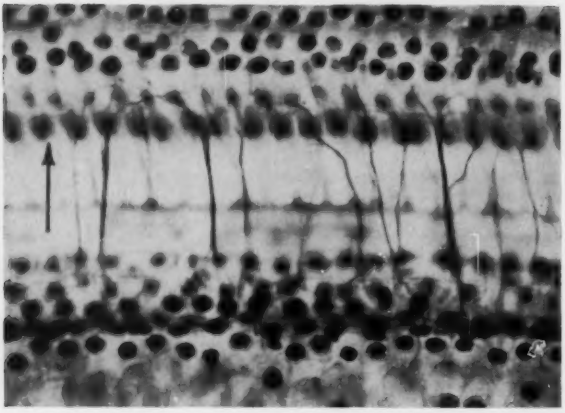

Fig. 10. Photomicrograph illustrating the fasciculi in the tunnel of Corti in the second turn. It shows several fasciculi of different density. Because of the thickness of the specimen many fasciculi and single fibres are out of focus. The arrow points to the outer hair cells. Bodian's method. 600×.

fibres in the tunnel of Corti in young guinea pigs was confirmed by Guild and for adult animals in the present studies. Toward the round window the medial fibres predominate while the basilar fibres are predominant toward the apex.

The central and peripheral myelinated processes of the ganglion cells have a diameter of 2.5μ to 3μ. Among the peripheral fibres there are many of small diameter which, in a few instances, were identified as collaterals of the intraganglionic spiral bundle. The central or peripheral processes were never found to exceed 3μ in diameter. The diameter of the unmyelinated fibres varied between less than 0.5μ and a maximum of 1.0μ. The smallest fibres were found in the internal spiral bundle, in the tunnel spiral bundle and among the basilar fibres crossing the tunnel to the outer hair cells. Those of greatest diameter were found in the internal spiral bundle and principally among the medial fibres which pass through the tunnel of Corti to the outer hair cells. There is no evidence of a systematic distribution of the fine and coarse

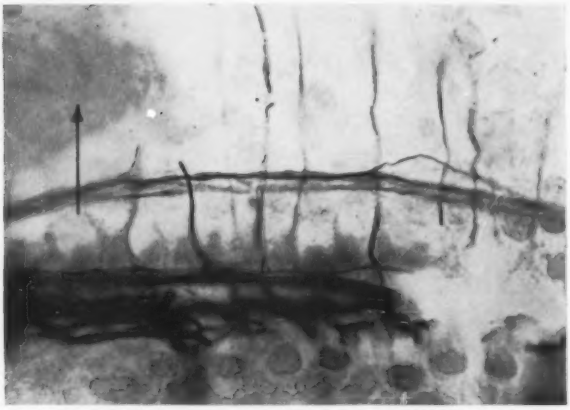

Fig. 11. Photomicrograph illustrating the tunnel spiral bundle in the apical turn. The arrow points to the outer hair cells. The bundle is formed by groups of few fibres crossing the tunnel on the upper part of the figure. To the left one fibre is turning out to the outer hair cells. In the lower part of the figure is the internal spiral bundle. Notice the great difference in size between the two bundles. The inner hair cells are out of focus. Single units or groups of two fibres are crossing to the outer hair cells. Cajal's pyridine method. 1260×.

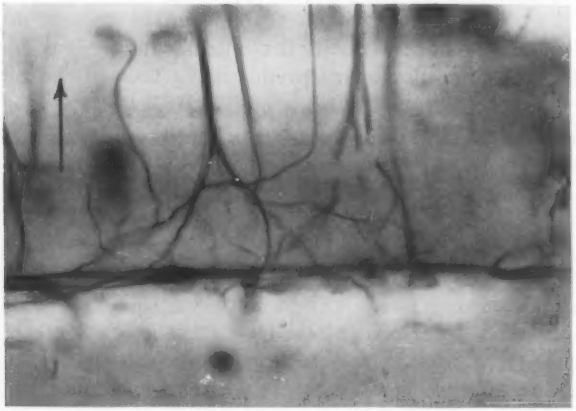

Fig. 12. Photomicrograph: the origin of the fasciculi in the tunnel of Corti in the first turn. It shows two fasciculi, each formed by many fibres coming from different places, and also, intermingled with them, single units and fasciculi of two or three fibres. In this area there are other fibres out of focus because of the thickness of the specimen. The arrow points to the outer hair cells. Bodian's method. 1400×.

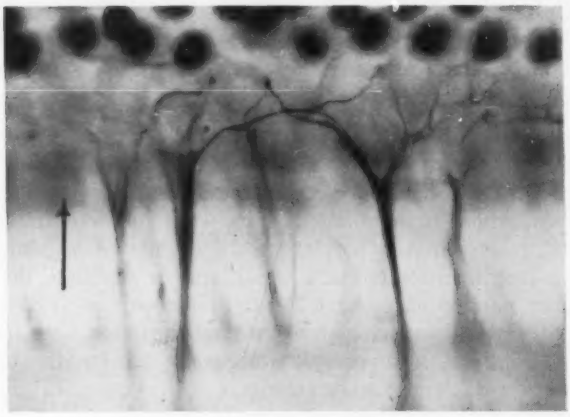

Fig. 13. Photomicrograph: the endings of the two fasciculi represented in Fig. 12 as they reach the outer hair cells. The fasciculi break up into individual fibres which take a spiral course toward either the apex or the round window. Bodian's method. 1260×.

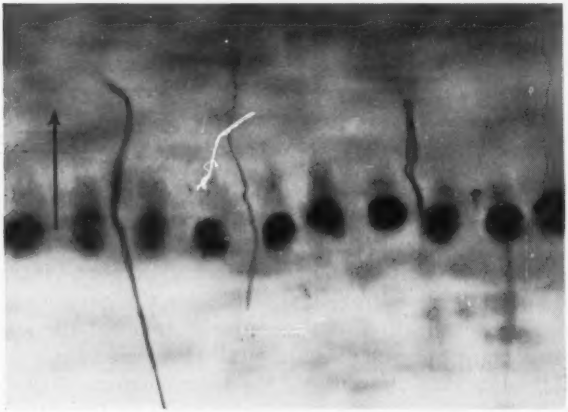

Fig. 14. Photomicrograph taken at the same place as Fig. 13 but focused at the foot of the outer pillars. It shows the nuclei of the outer pillar cells and single fibres or group of two fibres crossing between them to the outer hair cells. The arrow points to the outer hair cells. Bodian's method. 1260×.

fibres. In general, it may be said that toward the round window there is a predominance of coarse fibres while toward the apex the fine fibres are the most numerous.

The length of the fibres is extremely variable. The shortest are the radial fibres to the inner hair cells, whose myelinated processes measure about 1 mm. and the unmyelinated portions 30μ. The longest are the intraganglionic spiral fibres to the apex, whose unmyelinated parts were followed for about 300μ. The myelinated portions were calculated as about 10 mm. in length in Rosenthal's canal, and in addition, there is an initial portion of at least 10 mm. between the cochlea and the contralateral superior olivary complex. It has been impossible to establish with complete accuracy the lengths of the various fibres. The measurements are, however, in general agreement with the classical descriptions.

DISCUSSION.

In consideration of the discovery of Rasmussen the innervation of the cochlea should be regarded as formed by two different systems, namely, efferent and afferent.

The efferent system in the cochlea is the peripheral part of the tract of Rasmussen. The report of Rasmussen and the findings in animals with degenerative processes of the organ and ganglion of Corti have a triple interest:

a. Anatomically, the origin of the intraganglionic spiral bundle has been identified in the superior olivary complex of the opposite side. The cell bodies and dendritic processes are present in this nuclear mass and not in the ganglion of Corti as previously described. The possibility of some intraganglionic spiral fibres with cell bodies inside the ganglion is not denied. The localization of the nerve endings of the efferent fibres is a point to be clarified carefully because of the physiological implications. Lorente de Nó describes some fibres passing from the intraganglionic spiral bundle to the outer hair cells which the present studies confirm. The extent of this innervation on the outer hair cells is a question still to be answered.

Since the tract of Rasmussen enters the cochlea as a whole at the level of the first turn, and runs toward the apex, it is evident that some fibres in the intraganglionic spiral bundle are as long as the cochlea. The longest efferent fibre has the length of the tract of Rasmussen (to be determined in different animals) plus the intraganglionic course of the tract up to the apex (about 10 mm.), plus the spiral course underneath the inner hair cells (some of these were followed for 300μ). More important than the length is the ramifications of the efferent fibres. This is still to be determined.

b. Pathologically, it is of interest that atrophy of the primary neuron can occur in the presence of an almost normal intraganglionic spiral bundle. It is probably because the metabolic centers of the efferent fibres are far removed from the degenerative process. There is, however, a small reduction in the cross-sectional area of the bundle in the presence of atrophy of the primary neuron. It is not an artifact introduced by the plane of the section, because the difference, although small, is constant. This is a problem for further studies.

c. Physiologically, the fibres of the tract of Rasmussen presumably carry impulses from the superior olivary complex to the cochlea of the opposite side. The first suggestion of its function appears in the studies by Galambos *et al.*,[9] who found physiological evidence of a cochleocochlear interaction in the cat. As to whether this phenomenon is accomplished through the tract of Rasmussen, these authors state, "it is neither supported nor denied by the information at hand." The evidence for the efferent character of the tract of Rasmussen is at present almost entirely anatomical. The observations of Galambos *et al.* indicate rather strongly, however, that there are efferent fibres to the cochlea. In the absence of other known efferent fibres, this is significant circumstantial evidence supporting Rasmussen's anatomical interpretation.

It is remarkable that the size of the spiral bundle reduces suddenly at about the second turn. This fact suggests that whatever the function of these fibres may be, it is probably

more effective in the first turn. Since the first turn in the guinea pig is about one-half of the total length of the cochlea, the distribution of the terminal fibres of the tract of Rasmussen may have an anatomical rather than a physiological basis.

The afferent system is formed by two types of fibres, the radial and the spiral fibres.

To the radial fibres belong those which innervate a single or a small group of sensory cells. They are the radial fibres to the inner hair cells described by von Ebner and Lorente de Nó and the unbranched fibres to the outer hair cells described by Held. The radial fibres to the inner hair cells show a constant and systematic distribution throughout the cochlea while the radial fibres to the outer hair cells (unbranched fibres of Held) do not show a similar arrangement. Furthermore, the density of the unbranched fibres at different levels still is unknown. In any event, whatever the function of the radial fibres is, a principle of discrete localization exists for both types of sensory cells although it is more pronounced for the inner hair cells.

To the other type of afferent fibres, the spiral fibres, belongs the external spiral bundle with extensive ramifications to the outer hair cells. From the classical descriptions and the present observations, this is the predominant type of innervation on the outer hair cells. The functional significance of the diffuse innervation by the afferent spiral fibres is not known. Davis[10] suggests that it may be to allow spatial summation of weak local excitatory effects when the cochlea is stimulated by very faint sounds. It is doubtful that the inner hair cells have an afferent spiral innervation, since the internal spiral bundle, whose fibres give off numerous collaterals, is formed only by efferent fibres; therefore, the only afferent innervation of the inner hair cells is by the radial fibres. In other words the outer hair cells have principally a diffuse and overlapping innervation and the inner hair cells a more discrete or localized innervation. The inner hair cells are innervated by both the efferent and afferent systems while the outer hair cells are innervated almost exclusively by the afferent system.

SUMMARY.

The innervation of both the internal and the external hair cells is complex. In each case there are two classes of nerve fibres, the "radial" and the "spiral."

The radial fibres run directly outward from the spiral lamina and each fibre innervates one, two or, at most, three hair cells. The radial fibres form the chief nerve supply of the inner hair cells but they are in a minority in the supply of the external hair cells. Their cell bodies lie in the spiral ganglion (Corti).

The spiral fibres run for distances up to several millimeters in both directions along the organ of Corti. Each fibre innervates many sensory cells, although a given fibre does not innervate every hair cell along its course. A branch to every second or third cell is more typical. This type of innervation is predominant for the external hair cells. These spiral fibres form the well-known "external spiral bundles."

A given external hair cell may be innervated by more than one spiral fibre. A given inner hair cell may receive endings from sometimes one, usually two or, at most, three radial fibres. In addition some, at least, of the inner hair cells also receive endings from spiral fibres, and external hair cells receive some radial fibres in addition to their spiral fibres.

The spiral fibres to the external hair cells are afferent, with cell bodies in the spiral ganglion. The spiral fibres to the internal hair cells, however, seem to be *efferent* fibres. Their cell bodies lie in the medulla oblongata in the superior olivary complex of the *opposite* side. They run from this nucleus in the tract of Rasmussen and form a compact bundle in the auditory nerve. This bundle is clearly marked in the basal turn where it forms the intraganglionic spiral bundle. The collaterals terminate on internal hair cells. This bundle and the fibres of the tract of Rasmussen do not degenerate when the organ of Corti is destroyed by acoustic trauma.

The double innervation of the hair cells can be summarized as follows:

		TYPE OF INNERVATION	
		Dominant	Secondary
HAIR CELLS	Inner	RADIAL	SPIRAL
		Afferent	*Efferent*
	Outer	SPIRAL	RADIAL
		Afferent	Afferent

I express my appreciation to Dr. Walter P. Covell, Department of Otolaryngology, Washington University, for his technical advice and for furnishing the serial sections of animals traumatized acoustically; to Dr. Harold Schuknecht of the University of Chicago for furnishing me with his histopathological collection and for his criticisms; and to Dr. Stacy Guild for giving me the opportunity to examine his remarkable collection, for his criticisms and for his suggestions.

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PRIMARY CARCINOMA OF EXTERNAL AUDITORY CANAL, MIDDLE EAR AND MASTOID.*†

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INTRODUCTION.

Until recently, primary carcinoma of the external auditory canal, middle ear and mastoid has been considered as a medical curiosity. In reviewing the literature, I have found 211 authentic cases. It is quite possible that many more have been seen but have not been reported. Involvement of the auricle and external auditory canal are more apt to be seen by the dermatologist and radiologist but seldom by the otologist. According to statistics, the average practicing otolaryngologist will see one or, at the utmost, two cases of this distressing disease during a lifetime. I am fortunate in having seen my allotted number rather early in my career.

INCIDENCE.

The types of malignant tumors of this region run the gamut of new growths, ranging from hemangioendotheliomas to squamus cell and adenocarcinomas. The most rare are the adenocarcinomas, and Furstenberg,[6] Lukens[12] and Grabscheid[7] have reported cases of these. The squamus cell carcinoma is the one most frequently found. According to various authors, the frequency of occurrence varies from one in 5,000 to one in 20,000 of otologic cases. The condition is rare in persons under 40 years of age. It is equally distributed among the sexes. Grossman *et al.*[8] reported six cases, all in white females. The disease is exceptionally infrequent in the Negro race.

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ETIOLOGY.

The etiology of carcinoma of the ear is still obscure; however, certain associated and predisposing factors have been suggested as precursors to carcinoma. These are: *1.* chronic otorrhea, either from otitis media or mastoiditis; *2.* chronic eczema and other chronic dermatoses; *3.* senile keratoses; *4.* cutaneous horns; and *5.* chronic ulcerations from trauma or frostbite.

Although there is a wide diversity of opinion as to the relative significance of the various etiologic factors, most authors seem to stress the presence of a chronic aural discharge as a factor of great importance in carcinoma of the external canal and middle ear. Guttman,[9] on the other hand, asks "why we blame irritation of chronic discharge when there are so many chronic discharging ears that never become malignant." Figi and Hempstead[5] reported two cases of carcinoma following repeated attacks of erysipelas with ulceration of the external ear and canal. Danziger[4] reported cases in which "the condition was not preceded by chronic suppuration but occurred subsequent to acute otitis."

SYMPTOMATOLOGY.

The symptoms of carcinoma of the external auditory canal, middle ear and mastoid vary with the type, duration, size, location and invasiveness of the lesion. Basal cell carcinomas and adenocarcinomas of the mixed tumor type have aural symptoms of longest duration. Squamous cell carcinomas, especially of the middle ear, have the shortest duration of symptoms. The following are the most constant and frequent symptoms and findings that present themselves in this disease:

1. Chronic Aural Discharge: This is usually of many years' duration and is considered by Newhart,[15] Furstenberg[6] and Schall[21] to be of utmost significance when this discharge changes from a purulent to a bloody form. In the series of Figi and Hempstead[5] only 11 out of 38 cases had an aural discharge.

2. Pain: Pain in or about the ear is a very frequent complaint. It is severe and out of all proportion to the clinical findings. It usually follows the distribution of the trigeminal nerve and is sometimes aggravated by the opening and closing of the mouth. Furstenberg,[6] Schall[21] and Grossman *et al.*[8] believe that the pain is due to pressure of the growing tumor. Barnes[1] attributes the pain to infection of the tumor mass and skin lining the external auditory canal. Others favor the early invasion of the perichondrium and periosteum as the cause of this symptom. If the carcinoma arises in the middle ear or mastoid, the pain will present itself relatively early, but if it has its origin in the external auditory canal and invades the middle ear and mastoid secondarily, pain will be a very late symptom. According to Shuster,[22] the presence of intense pain suggests a malignancy, since a chronically discharging ear does not usually have pain.

3. Aural Growths: Occasionally a patient may seek medical advice because of this symptom alone. It is often very difficult clinically to differentiate between aural polyps, granulations and a malignancy. Schall,[21] Lederer[11] and other authors stress the suggestiveness of malignancy in any polyp or granulation tissue that bleeds profusely when removed or manipulated.

4. Ulceration: Ulcerations of the external auditory canal which persist and become worse in spite of treatment are very suggestive of an underlying neoplasm.

5. Palsies of the Various Cranial Nerves: Buckman[3] states that facial paralysis occurs frequently enough to be considered a symptom of carcinoma. When this is seen, the lesion is usually far advanced. Occasionally the IXth, Xth, XIth and XIIth nerves may become involved by the neoplasm, giving a jugular foramen syndrome with dysphagia, inarticulate speech and hoarseness. With invasion of the petrous pyramid there may at times be a paralysis of the IIIrd and VIth nerves.

6. Deafness and vertigo are infrequent symptoms. The deafness is usually proportionate to the amount of obstruction of the canal and involvement of the ossicular chain.

DIAGNOSIS.

"Successful treatment of carcinoma, regardless of its location, depends on early diagnosis. This is especially true in the middle ear, for by the time the lesion is grossly visible, invasion of the underlying bone has often already occurred" (House[10]). A diagnosis of suspected malignancy can be made from a careful history, the symptoms and significant clinical findings. A biopsy, however, is essential and, if negative, should be repeated as often as feasible. House[10] suggests the use of the Papanicolaou technique in all chronically discharging ears and also where a biopsy is not possible.

X-ray examination of the temporal bone is of little value in the early diagnosis of carcinoma; however, it is of inestimable value in determining the extension and bony involvement by the disease — a factor which is very important if surgery is contemplated.

PATHOLOGY.

Several authors have attempted to classify carcinoma of the ear into extrinsic and intrinsic forms. Lederer[11] has suggested the tympanic membrane as a natural anatomical delineation for clarifying these lesions of the ear. Schall[21] states that "a neoplasm in the external auditory canal beyond the isthmus is a potential malignant growth of the middle ear."

Metastasis of carcinoma of the ear to distant body organs is rare. Regional lymph nodes are often enlarged, but sections of them usually reveal a chronic inflammatory reaction. The disease is dangerous mainly because of its position and tendency to invade adjacent vital structures. The dura and the labyrinthine capsule are uniquely resistant to invasion by the neoplasm until very late in the course of the disease. House,[10] Towson and Shofstall[24] have reported cases of intracranial extension. Erosion of the internal carotid with fatal hemorrhage has been observed. Cranial nerve involvement other than the facial has been seen by several authors. Extension to the temporomandibular joint and parotid gland occurs relatively early.

A number of authors stress the fact that the lining membrane of the middle ear will undergo a change, with a change in the character of the suppuration. Panse[16] states "that a neoplasm which originates in a closed tympanic cavity probably results from a metaplasia of the cylindric cells of the lining membrane to the squamous cell type or has its origin from the glandular elements within the cavity."

TREATMENT.

The consensus of the opinion among all the authors is to treat this distressing disease with radical surgery, where feasible, and irradiation. Figi and Hempstead[5] report good results in their series, using electrosurgery and intensive irradiation by radium points directly to the site of the lesion. Others have used radical surgery and external Roentgen radiation. Schall,[21] in 1935, pointed out that "in the external use of irradiation, the clinical fact is ignored that with a dosage sufficient to cause necrosis of the bone, it may have no effect on the tumor invading the bone." He also suggested the following procedures:

1. Excision of the entire cutaneous canal.
2. Radical mastoidectomy for the purpose of inspection and for sufficient space for proper application of radium.
3. Irradiation must be applied at the site of the lesion.

Post-treatment follow-ups should be done at frequent intervals. Papanicolaou smears may indicate the progress of the disease.

PROGNOSIS.

According to Figi and Hempstead,[5] the prognosis of carcinoma of the external auditory canal, middle ear and mastoid is comparable to malignancy of the paranasal sinuses. In Schall's[21] series of five cases there was no recurrence in two to four years, but at the end of seven years all were dead except one, a hemangioma. In the series of 38 cases of Figi

and Hempstead,[5] 20 patients lived two years, 15 lived three years, 10 lived seven years, eight lived eight years, three lived 10 years, and one lived 15 years.

CASE REPORTS.

I wish to present briefly five previously unreported cases of squamous cell carcinoma of the external auditory canal, middle ear and mastoid from the files of the University Hospitals. I have had personal contact with Cases 1 and 2 only.

Case 1: Mrs. M. D., a white female, age 56, came to the O. P. D. on Aug. 25, 1950, because of itching (five years' duration) and progressive deafness (two years' duration) of the right ear. There was no aural discharge at any time. Examination revealed a severe desquamating external otitis of the right ear with a pale red mass occluding the lumen of approximately the inner half of the external auditory canal. This mass bled easily when manipulated. There was tenderness in the right temporomandibular joint on palpation and pain on opening and closing of the mouth. Biopsy was deferred until the external otitis was under control. On Sept. 14, 1950, a biopsy of the mass was taken and was reported as a well differentiated squamous cell carcinoma.

The patient was admitted into the hospital on Sept. 16, 1950. Physical examination again revealed the above findings and also a bilateral mixed type deafness, worse on the right ear, a ventral hernia, and a mild hypertension. Laboratory findings were within normal limits. The serology was negative.

The X-ray report on the day of admission was "essentially negative examination of the mastoid regions with slight diminution and radiolucency of the right mastoid cells and no evidence of bone destruction seen" (see Fig. 1).

On Sept. 20, 1950, an endaural radical mastoidectomy was performed on the right ear. The external auditory canal was excised, completely removing thereby the major portion of the tumor. A small amount of soft friable tissue was found in the hypotympanum, and there seemed to be some invasion of the tympanic plate and floor of the external auditory canal. The remainder of the middle ear, antrum and mastoid were relatively free of tumor. Preauricular nodes were not removed. The cavity was not skin grafted.

The pathological report of the tissues removed at surgery was well differentiated squamous cell carcinoma clinically of the external auditory canal and in bone (see Figs. 2A and 2B).

On Oct. 6, 1950, the patient was started on external Roentgen therapy. She received a total of 3,600 r. through one field. A herniarrhaphy was done on Nov. 1, 1950, and she was discharged on Nov. 9, 1950.

She was seen in the O. P. D. twice a week. Papanicolaou smears were taken every two weeks. So far all have returned as negative for malignant cells. The cavity is epithelializing nicely at the present time.

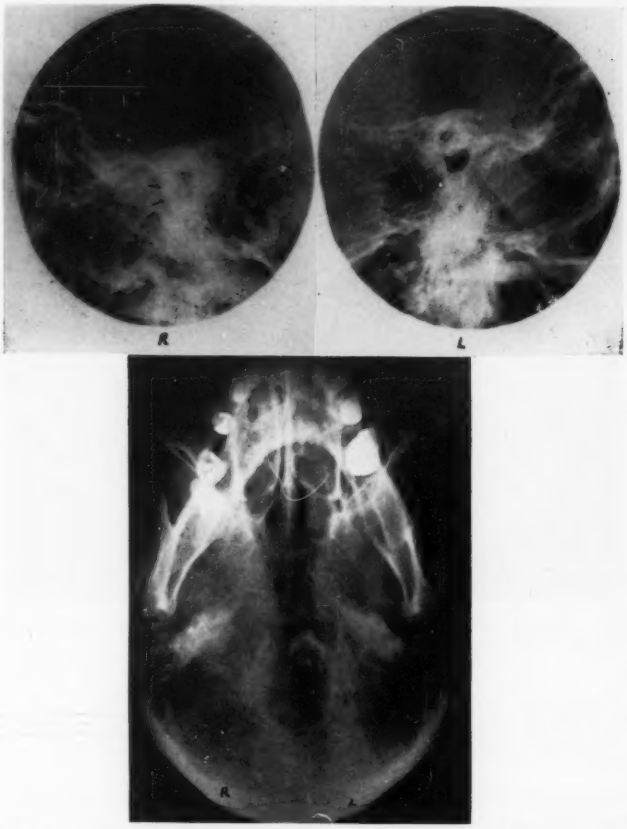

Fig. 1. Case 1. Roentgenograms of both mastoids and petrous pyramids showing no bone invasion. Arrows indicate approximate areas where bone invasion was found during operation.

Case 2: Mr. O. K., a white male, age 72, was admitted into the hospital on Nov. 8, 1950, with a complaint of chronic discharge from the right ear (35 years' duration) and severe pain in the right side of the face (one month's duration). The pain radiated to the temporal region, zygoma and mandible. It was aggravated by opening and closing of the mouth. He had an "abscess" of the right ear on Oct. 24, 1950, with a bloody discharge. Physical examination revealed the right external auditory canal almost completely filled with a pink granular mass. The drum was not visualized. The left ear, except for a very narrow canal and a small

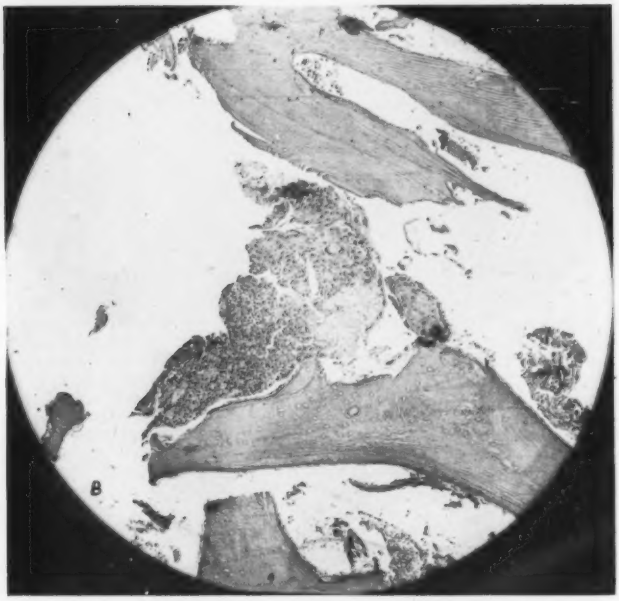

Fig. 2. Case 1. Biopsy of right external auditory canal showing well differentiated squamous cell carcinoma.

central perforation, was normal. There were no palpable regional nodes. He also had a moderate right facial weakness, a deformity of the right leg and foot, a mild hypertension and a left inguinal hernia.

A biopsy of the mass in the right external auditory canal was done on Nov. 9, 1950, and revealed a well differentiated squamous cell carcinoma (see Fig. 4).

Roentgen examination of both mastoids and petrous pyramids on Nov. 9, 1950, was reported as "there appears to be invasion and possibly erosion of the tympanic portion of the right ear by a carcinoma which is known to be present clinically. There is also mottling of the ear extending towards the tip of the petrous pyramid, probably due to invasion here as well" (see Fig. 3).

The laboratory findings were within normal limits. Due to the patient's age and X-ray evidence of extensive invasion, it was deemed inadvisable to perform any surgery. He was immediately started on external Roentgen therapy. He received a total of 3,500 r. through one field.

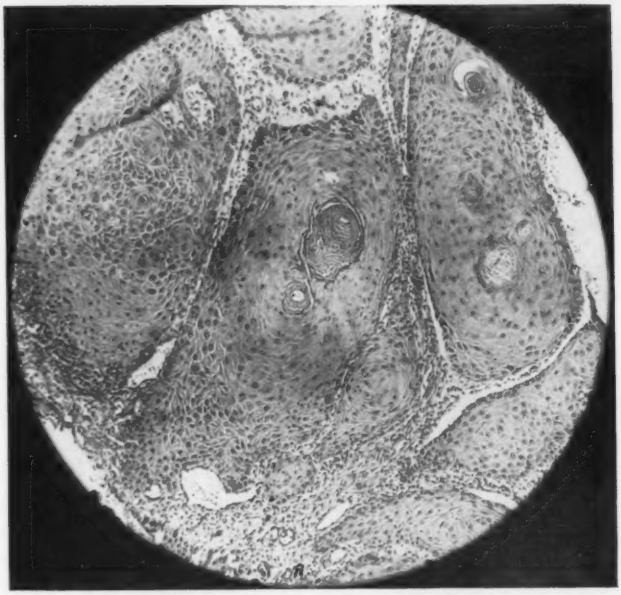

Fig. 2. Case 1A. Curetted biopsy from the right tympanic plate showing the carcinoma invading the bone.

A follow-up on Jan. 10, 1951, showed the canal dry and smooth, but markedly narrowed towards the drum. The drum was not visualized. The patient stated that he had some relief from the intense pain after the irradiation.

Case 3: Mr. J. B., a white male, age 31, was admitted to the hospital on March 31, 1939, with a complaint of a discharging left ear since the age of two years, severe pain in that ear and a growth in the canal of the left ear of five months' duration. Prior to admission he had four attempts at removal of the mass for the relief of pain. Two weeks prior to admission a fifth biopsy was done and revealed the mass to be carcinoma.

Physical examination showed a purulent discharge in the left external auditory canal and a red mass at about the junction of the bony and cartilaginous portions of the canal. The drum was not visualized. There was no hearing by air, and bone conduction was poor. The right ear was normal.

On April 1, 1939, the Roentgenographic report was a "malignant tumor originating in the left external auditory canal with considerable amount

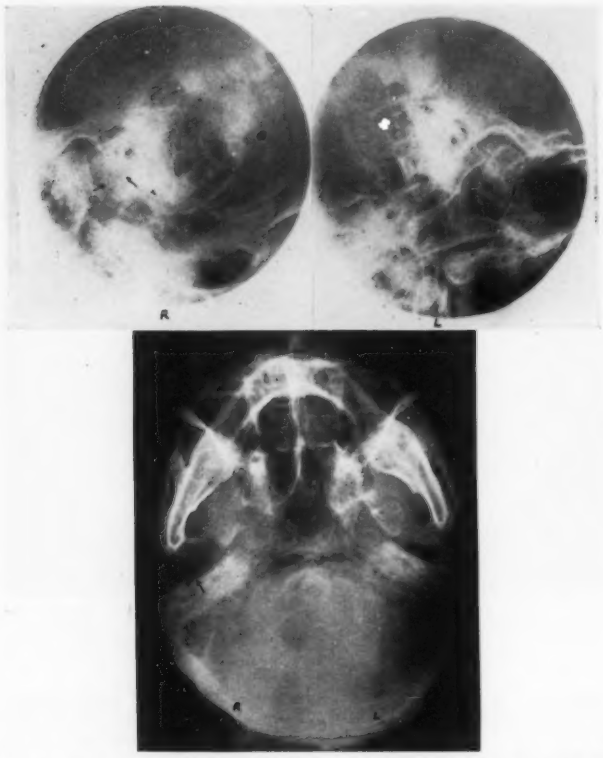

Fig. 3. Case 2. Roentgenograms of both mastoids and petrous pyramids showing invasion and erosion of the tympanic portion of the right ear and also a mottling of the bone extending towards the tip of the petrous pryamid.

of bony destruction around it. In left mastoid, cells are absent. In the region of the auditory canal there is an extensive area of bone destruction which measures 2 cm. by 1.5 cm. The area of bone destruction in the petrous bone is secondary to the tumor" (see Fig. 5).

A postauricular radical mastoidectomy with excision of the membranous external auditory canal was done on April 1, 1939. The major portion of the tumor was removed with the membranous canal. The middle ear was free of tumor tissue. "It was felt at the time of operation that the tumor tissues extended down to the middle ear, possibly invading the left temporomandibular joint."

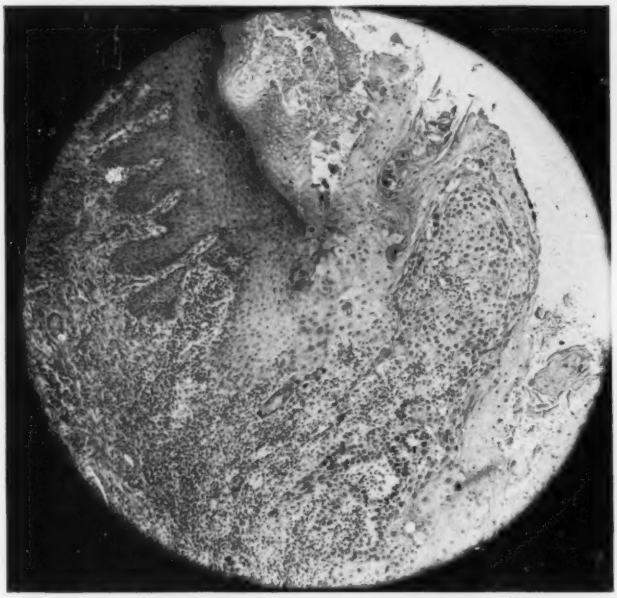

Fig. 4. Case 2. A biopsy of the tumor in the right external auditory canal showing a well differentiated squamous cell carcinoma.

The pathological report on the tissue removed at surgery was "highly undifferentiated squamous cell carcinoma."

The wound was left open and the patient received external Roentgen therapy directly to the site of the lesion. In one series, using one field and a 60 K. V. machine, he received a total of 4,400 r. In a second series, using two fields and a 220 K. V. machine, he received a total of 4,437 r.

One year after irradiation, bony sequestra were removed from the canal.

The cavity and the canal epithelialized. The patient is at present alive and in apparent good health, 12 years after surgery and irradiation. From personal communications with the attending physician, I gather that there is no evidence of a recurrence to date.

Case 4: Mrs. A. D., a white female, age 69, was admitted into the hospital on Sept. 17, 1948. One year prior to admission the patient felt a "wart" in her right ear. She continually would pick at it with her fingers as she was constantly aware of its crusty presence. There were no other

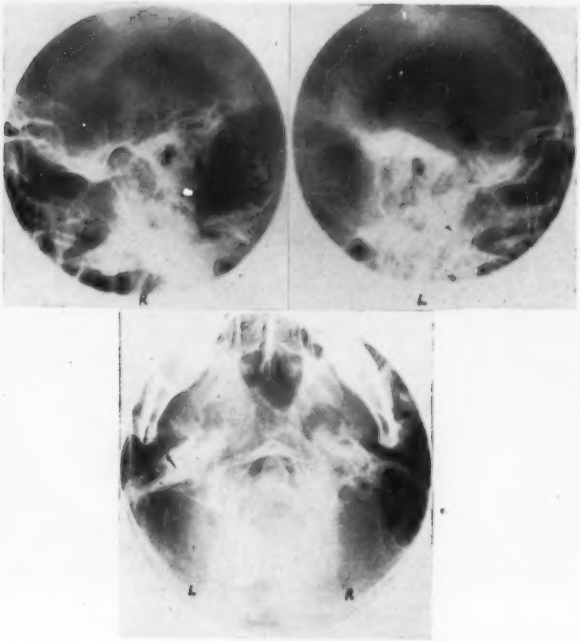

Fig. 5. Case 3. Roentgenograms of both mastoids and petrous pyramids showing extensive bony destruction in the left external auditory canal, mastoid and petrous pyramid.

symptoms until about five months prior to admission. At that time she began to have pain, occasional bleeding from the canal, and difficulty in chewing because of pain in the right temporomandibular joint. She also complained of swelling behind and anterior to the right ear. A local doctor made sections of the "wart" on five occasions with no pathological report. Another doctor took a biopsy and reported it as "radioresistant carcinoma." The month prior to admission the patient had lost weight and had experienced severe and constant vertigo except when absolutely quiet.

Physical examination revealed a horizontal nystagmus on right lateral gaze. The right ear was tender, the mastoid area was swollen, the auricle protruded and was tender when pulled, and there was a large movable node anterior to the ear. There was a large, soft, pink smooth mass filling the right external canal. The tissue bled easily, and there was considerable purulent exudate in the canal. The left ear was normal.

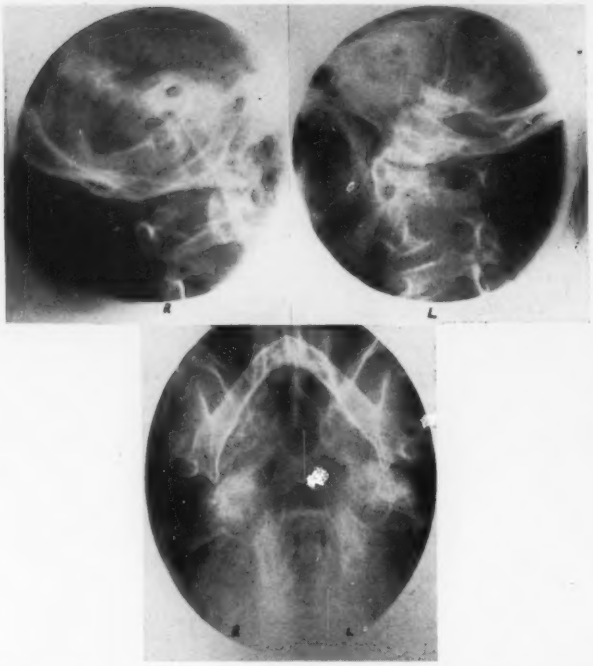

Fig. 6. Case 4. Roentgenograms of both mastoids and petrous pyramids showing extensive bone destruction involving the mastoid process of the right temporal bone, extending forward to uncover the right temporomandibular articulation and medially to destroy the lateral portion of the right petrous ridge.

There was no demonstrable hearing in the right ear. The rest of the physical examination was essentially normal except for varicose veins and discolorations of both legs.

The X-ray examination of the mastoids and petrous pyramids on Sept. 17, 1948, was reported as "extensive bone destruction involving the mastoid process of the right temporal bone, extending forward to uncover the right temporomandibular articulation and medially to destroy the lateral portion of the right petrous ridge" (see Fig. 6).

On Sept. 20, 1948, the major portion of the tumor mass and the right external auditory meatus were excised as a biopsy. The pathological report was "partially differentiated squamous cell carcinoma clinically of the external auditory canal."

A right preauricular abscess was incised and drained on Sept. 24, 1948, and she received a total of 4,437 r. through two fields.

After discharge from the hospital the patient was not seen again in either the O. P. D. or in any other department. The outcome of this case is, therefore, unknown.

Case 5: Mr. J. M., a white male, age 39, was admitted into the hospital on Aug. 11, 1940, with a complaint of severe and constant pain in the left ear of nine weeks' duration. At the onset of his symptoms he was seen by a physician who told him that he had a pimple in his ear and treatment was given. The pain was not relieved. He was again seen by his physician and at this time some bone was found to be exposed in the left external auditory canal. The patient received treatment for this condition, including some X-ray therapy, but without relief of the pain. The patient then had three molars extracted, a tonsillectomy and treatment for a sinus infection. He was finally referred to a local physician who took a biopsy of the lesion, which was diagnosed as a well differentiated squamous cell carcinoma.

The physical examination on the day of admission was essentially normal except for the left ear, which showed a very tender, red, swollen external auditory canal. There was a small area of bone visualized in the anterior canal wall and also along the floor of the canal. The entire membranous portion of the canal up to the drum seemed to be involved. Laboratory findings were within normal limits. Serology was negative. X-rays showed catarrhal mastoiditis on the left. On the day following admission the entire membranous portion of the external auditory canal was excised through a postauricular incision. The drum was left intact. Electric cautery was then applied to the remaining tissue of the canal.

The patient subsequently received a series of X-ray treatments at St. John's Hospital.

On Aug. 25, 1940, he was again admitted to this hospital because of a recurrence of his symptoms and the carcinoma. At this time a small, firm mass was noted just below the lobule of the left ear. This mass was adherent to the overlying skin. It was the opinion of the patient's physician that this was a metastatic lesion and that radical surgery at this time was inadvisable and the patient was discharged.

The patient subsequently died.

CONCLUSION.

The literature concerning carcinoma of the external auditory canal, middle ear and mastoid has been briefly reviewed.

Five new cases of squamous cell carcinoma of this region are presented.

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THE TONSILLECTOMY-POLIOMYELITIS PROBLEM. A REVIEW OF THE LITERATURE.*†

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Some progress has been made toward a better understanding of the tonsillectomy-poliomyelitis problem but sharp basic differences of opinion still exist despite the vast medical literature published on this subject.

Sheppard, in 1910, was the first to study the relationship of poliomyelitis and tonsillectomy.

In 1928, Ayer reported nine cases of bulbar poliomyelitis following tonsillectomy by a five to 10-day interval.

Aycock and Luther,[1] in 1929, reported a controlled study of 714 cases of poliomyelitis which revealed an excessive number of bulbar cases following tonsillectomy after a specific time interval. Of 217 cases (30 per cent) who had prior tonsillectomy, 16 had been operated seven to 18 days preceding the onset of the disease and all of these 16 cases were of the bulbar type.

Eley and Flake,[2] in 1938, studied 418 consecutive cases admitted to infants' and children's hospitals of Boston. Of the 287 spinal cases, eight (2.7 per cent) had been tonsillectomized seven to 30 days previously. Of the 131 bulbar cases, 17 (13 per cent) had tonsillectomy between the tenth and twentieth days preceding onset of the illness.

Stillerman,[3] in 1938, analyzed 686 cases of poliomyelitis admitted to Willard Parker Hospital during 1935 and reported

*In this paper, tonsillectomy refers to either tonsillectomy, adenoidectomy or both.

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13 cases of poliomyelitis, nine of which were bulboencephalitic, developing 10 to 22 days after adenotonsillectomy.

The report of Krill and Toomey,[4] in 1941 on the K family, of Akron, Ohio, was followed by widespread publicity in both lay and medical publications. In the K family tonsillectomy was performed August 22 on five apparently healthy children. In less than three weeks all had developed bulbar poliomyelitis and three had died. The sixth and youngest child was not operated and remain well, though the virus of poliomyelitis was recovered from his stool. Only two scattered cases of polio were reported in Akron prior to the K family cases.

Francis, Krill, Toomey and Mack[5] studied these K family cases and concluded that the operative procedure precipitated the severe bulbar form of poliomyelitis in children who otherwise probably would have escaped with inapparent infections.

Cunning,[6] in 1949, concluded: "After 10 years' study of this subject, following over 17,000 poliomyelitis cases and 35,000 tonsillectomy cases, I am still of the same opinion that I was one year ago; namely, that no definite causal relationship between tonsillectomy and poliomyelitis has been established; therefore, I do not believe that tonsillectomy should be postponed indefinitely simply because the summer months are the months during which poliomyelitis is prevalent. If, however, there is a distinct rise in the poliomyelitis rate, bordering on epidemic proportions, in any community, all elective operations should be postponed.

"Until evidence of a conclusive nature is forthcoming, the surgeon should decide whether or not to perform tonsillectomies during the summer months and whether the dangers of delaying the operation are greater than the still undetermined risk of increasing susceptibility to poliomyelitis."

Faber[7] re-examined Cunning's 1946 and 1947 reports and found the incidence of bulbar cases more than twice as great in patients tonsillectomized within two months of onset as in nontonsillectomized patients.

Fischer, Stillerman and Marks,[8] in reporting the 1937 Toronto epidemic, concluded that among children between the ages of three and 12 years, acute poliomyelitis developed more often in those recently tonsillectomized than in others and that the increased incidence was due entirely to the excess of bulbar cases. Among the children with poliomyelitis, they found the incidence of the bulbar form more than twice as great in those whose tonsils had been removed at any time previously as in those with tonsils intact.

Toomey and Krill,[9] in reporting cases over a 14-year period at Children's Hospital, Akron, and one year (1941) at City Hospital, Cleveland, stated there were more individuals who had had tonsillectomies and adenoidectomies within 30 days prior to the onset of poliomyelitis who developed a bulbar type of the disease than could be explained on the basis of mere chance or random sampling.

Lucchesi,[10] in 1944, analyzed 432 cases of acute anterior poliomyelitis and found the incidence of bulbar and bulbospinal poliomyelitis was significantly higher in patients without than in patients with tonsils and adenoids.

John Anderson[11] studied the 1943 Utah epidemic and reported that 43 per cent of the bulbar and bulbospinal cases were preceded by a tonsillectomy within 30 days of the onset. The incidence of poliomyelitis was 2.6 times greater and the incidence of the bulbar and bulbospinal type 16 times greater in recently tonsillectomized children than in the general child population.

Campbell,[12] in analyzing 303 patients admitted to the Colorado General Hospital during the 1946 epidemic, found that previous tonsillectomy seemed to increase the individual's susceptibility, particularly children, to the bulbar type if poliomyelitis was contracted.

Roberts,[13] in 1946, summarized his views on 274 cases of poliomyelitis following recent tonsillectomy during the last 35 years, stating that the annual incidence of poliomyelitis in everyday life is 31 times greater than in the recently tonsillectomized population even during the poliomyelitis season.

Pederson,[14] in 1947, studied 492 cases of poliomyelitis in San Francisco (1941-1945) and reported 11 cases following tonsillectomy. He found the incidence of poliomyelitis following recent tonsillectomy was not greatly out of proportion to the ratio of disease to the general population during an epidemic year, but that when poliomyelitis did occur following tonsillectomy it was more apt to be bulbar in type. He also concluded that there was a two to one higher incidence of bulbar poliomyelitis in tonsillectomized than in nontonsillectomized patients.

Winborn and Stansbury[15] made a survey of 134 poliomyelitis cases admitted to Parkland Hospital during the first eight months of 1946. Their conclusions were that the incidence of poliomyelitis in tonsillectomized individuals is no greater than in those individuals whose tonsils have not been removed and the danger of contracting poliomyelitis following recent tonsillectomy is minimal.

Eschenbrenner,[16] in 1948, reviewed the literature, stating that the incidence of poliomyelitis probably is not appreciably greater following tonsillectomy, but certainly when a tonsillectomy patient develops poliomyelitis soon after operation, it is usually bulbar in type.

Kinney[17] surveyed 237 cases of poliomyelitis occurring in Cuyahoga County, Ohio, in 1946 and 1947. He stated that the relationship between tonsillectomy and the acquiring of polio is no more than casual and the public hysteria concerning this problem is unwarranted.

Wesselhoeft[18] formerly favored a ban on tonsillectomies during the summer months but has revised his opinion and now recommends that the operation be performed immediately after the close of school in June in Massachusetts.

In a personal communication, Dr. Wesselhoeft[19] states that in Massachusetts the State Board of Health furnished all hospitals with a bulletin every week during the summer giving the number and location of cases and makes a statement as to whether or not it is advisable to refrain from tonsillectomies.

Later in a city where it had been considered wise to refrain from tonsillectomies, the hospitals are advised to resume operations when the case rate has fallen in that locality.

Dr. Feemster,[20] Director of the Division of Communicable Diseases for the Massachusetts Department of Public Health, in a personal communication described the origin and method of operation of the above noted Massachusetts procedure which has given the hospitals a uniform policy, helped reduce confusion of the public and rendered the position of the otolaryngologist more comfortable.

Gaylord Anderson,[21] in 1950, studied the 1946 outbreak of poliomyelitis in Minnesota and concluded that the risk of developing poliomyelitis was at least three times as great among those undergoing tonsillectomy as among a comparable group not undergoing this operation and the risk of bulbar infection was 11 times as great.

Treynor,[22] writing in 1950 of the 1948-1949 epidemic in southwestern Iowa, could demonstrate no connection between tonsillectomy and poliomyelitis, spinal or bulbar, in the 374 hospital cases studied.

Miller,[23] at the American Medical Association meeting in 1950, reported on 1,229 cases occurring in Los Angeles County in 1949. The results of his analysis indicated no clinical or statistical significant deviation between the actual and the expected incidence of poliomyelitis developing in Los Angeles County during 1949 in patients recently tonsillectomized even in the months of July through October. He concluded there was no reason found to discontinue adenotonsillectomies during the summer months.

Goerke[24] (personal communication) believes the rate of bulbar involvement of poliomyelitis cases is significantly higher following adenotonsillectomy than in cases not following these operations and is convinced that it is a great responsibility on the individual physician to perform an elective adenotonsillectomy during the summer months in Los Angeles. He

feels that deferment of elective adenotonsillectomy is wise during the increased incidence of poliomyelitis, which in the city of Los Angeles is from June through November.

The Texas, 1950, poliomyelitis cases totaled 2,778 and the peak month was July with 571 cases. A total of 1,492 Epidemiological Report Forms were received by the Division on Epidemiology. Of this number, 42 answered yes in regard to recent (three weeks) tonsillectomy or dental operation, but this question was not answered on 28 of the forms. The bulbar cases totaled 91 and five of these were preceded by recent tonsillectomy or dental operation.

Greenberg[25] (personal communication) states that in his analysis of the 1949 epidemic in New York City the actual number of cases of poliomyelitis occurring within one month after operation was three and one-half times the expected number. Also, for the years 1944 to 1949 the percentage of cases with bulbar paralysis within one month of operation was 17 times greater than in a control group of nontonsillectomized children.

Aycock,[28] in 1942, reviewed 170 cases of poliomyelitis following tonsillectomy within 30 days and 121 (71 per cent) of these were either bulbar or bulbospinal in type. In 1949, Aycock presented to the Eastern Section of the American Laryngological Society a paper[27] which is worthwhile reading to obtain his well expressed attitude toward tonsillectomy and poliomyelitis, but a synopsis here would not do the paper justice. Some of Aycock's views have been incorporated in the following comment and conclusions.

COMMENT AND CONCLUSIONS.

There is unmistakable evidence that the attack rate of bulbar poliomyelitis in many epidemics has been found to be significantly higher in recently tonsillectomized patients, and, evidence is increasing that bulbar poliomyelitis rather than the spinal form of the disease is more likely to occur in patients tonsillectomized at any time than in those with tonsils intact.

This reviewer agrees with Aycock's statement that the fact which proves beyond a doubt that there is a relationship between tonsillectomy and poliomyelitis is the greater frequency with which bulbar poliomyelitis is seen to follow tonsillectomy by a specific interval. Other types of poliomyelitis do not follow tonsillectomy by this specific time interval.

The danger of bulbar poliomyelitis following tonsillectomy is almost zero except during periods of increased incidence or epidemics when, although the risk is not great, it is definite and real and increases in proportion to the increase in incidence. Since the hazard of the tonsillectomy-bulbar poliomyelitis sequence is small, it should not preclude adenotonsillectomy for definite medical indications, but at the same time it is advisable to take into consideration the timing of the operation, which is elective in most cases.

Adenotonsillectomies should not be performed during an epidemic or on patients with known recent exposure to poliomyelitis, and, the operation should be delayed when there is an increased incidence of poliomyelitis in the community unless postponement jeopardizes the patient's health to a greater degree.

The practice of the Boston hospitals appears logical. These hospitals follow a uniform procedure agreed upon by them and based on information and recommendations furnished by their State Board of Health relative to increase in prevalence of the disease in any of the various areas served by the hospitals.

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DIHYDROSTREPTOMYCIN-BORIC ACID POWDER IN THE TREATMENT OF AURAL DISCHARGE.

A CLINICAL REPORT.*†

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Notwithstanding the advances and modifications that have occurred in the practice of otolaryngology in the past 15 years, the problem of aural discharge remains one of the most common to confront the otologist in his daily office routine. During these years many new drugs have come to the treatment table, some to be discarded, others to find a permanent place in our armamentarium. The purpose of the present report is to record clinical experiences with one of the new antibiotics—dihydrostreptomycin—used in conjunction with an acid, drying agent—boric acid—and to present biochemical and bacteriological data that may offer some explanation of the beneficial results observed in the clinical use of this mixture.

REVIEW OF THE LITERATURE.

Comprehensive and repeated studies of the bacteriology of aural discharge have been reported in recent years and it does not seem necessary to include well established data; however, a review of the literature directly concerned with the present report seems pertinent.

Collins and Hughes[1] studied the bacterial flora occurring in 26 ears of 23, previously untreated patients with chronic

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otitis media, and they observed that Gram-negative bacilli were more frequently present than Gram-positive cocci. Repeated cultures of flora from the middle ear cavities of certain patients treated with local penicillin revealed a complete change from the original, predominantly Gram-positive organisms—sensitive to penicillin—to Gram-negative bacilli—resistant to penicillin. They concluded that, "The presence of the secondary organisms, such as the 'coliform' bacilli, B. proteus and diphtheroids was undoubtedly one of the complicating factors limiting the successful treatment of chronic suppurative otitis media by penicillin."

Pseudomonas aeruginosa was the predominant organism in 45 of 100 consecutive cases reported by Salvin and Lewis.[2] In another study[3] of 72 ears, the same organism was found in 67 per cent of the cases. Other Gram-negative bacilli were cultured in 17 per cent. These organisms were also noted in other cases of otitis externa, and the failure of local and parenteral penicillin therapy has been ascribed to their presence in the exudate.[4]

Holt and Snell[5] reported 20 cases of chronic otitis media in which cultures from 26 ears had been made. *Pseudomonas aeruginosa* was present in 12 instances and *Bacillus proteus* in 14 cultures.

Senturia[6] states, "In a recent report on 135 cases of external otitis observed at Randolph Field, Tex., and in reports from New Guinea and the Solomon Islands, it was shown that fungi were found in a small number of cases and that Gram-negative bacilli (especially the Pseudomonas group) were found in a very large percentage of cases observed in these tropical and subtropical areas."

Douglas,[7] in an analysis of the bacteriology of 142 ears, reported the prevailing organisms to be "Staphylococcus, Proteus, Pseudomonas and Streptococcus" in the order named.

Hayes and Hall,[8] in a clinical and bacteriological study of 160 cultures from 100 cases, reported that the resistant etio-

logical agent in chronic suppurative otitis media and external otitis was predominantly *Pseudomonas aeruginosa.*

Gill[9] presented results of 252 cultural studies of the external ears of 168 patients and reported that bacteria were present in 117 cases, bacteria and fungi in 48, and fungi alone in three cases. Gram-negative bacilli occurred most frequently (104 cultures). Staphylococci were present in 96 cultures.

All of these authors have stressed the importance of using an agent that is effective against Gram-negative bacilli in the management of aural discharge.

The effect of vehicles on the antibacterial action of various substances was extensively studied by Senturia and Doubly.[10] Using the cultures of *Staphylococcus aureus* and *Pseudomonas aeruginosa,* they found the action of streptomycin to be unimpaired by such vehicles as distilled water, glycerin, Carbowax and "Carboprop." On the other hand, the effectiveness of streptomycin was completely inhibited when the vehicle used was lanolin, Aquaphor or Hydrosorb, all water-insoluble vehicles.

The susceptibility to infection may be due to such predisposing factors as moisture together with maceration of the skin, absence of cerumen, and the accumulation of dead, keratinized, horny tissue with the neutralization of the antibacterial, acid cloak.

The pH of the cerumen and of the skin of the canal, which is normally acid, is of special importance. In healthy ears, the pH is within the acid range. Fabricant and Perlstein[11] studied the pH of the external ear in 27 infants, 44 children and sixty adults with no apparent lesions in their external auditory canals, and found that pH values fell within the acid range in the majority of instances (pH range: 5.0 to 7.8). They conclude, "The management of the more difficult and more resistant cutaneous infections can be improved by a better understanding of the manner in which the skin resists and harbors bacteria and of some of the mechanisms by which bacteria produce harmful effects. Several factors play a rôle

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
1	Itching, deafness	Unknown	Diffuse otitis externa	5	Improved
2	Itching and crusted ears	5 years	Diffuse crusting	11	Allergic dermatitis
3	Discharging left ear	1 week	Diffuse purulent otitis externa	1	Not known
4	Painful and swollen left ear	several days	Unlocalized furuncle	13	Improved
5	Painful ear	several weeks	Diffuse otitis externa with bloody blebs	10	Improved
6	Discharging left ear	1½ years	Diffuse otitis externa	1	Not known
7	Itching and crusted ears	1 year	Diffuse otitis externa with crusting	4	Improved
8	Irritation and itching	several weeks	Mild otitis externa	1	Improved
9	Itching and discharging ears	2 months	Diffuse otitis externa with crusting	5	Improved

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
10	Painful, itching, and discharging ears	2 years	Severe bilateral diffuse otitis externa	4	Improved
11	Painful crusted ear and face	Unknown	Crusting dermatitis	1	Not known
12	Itching, crusted, discharging ears	4-6 months	Neuro-dermatitis?	9	Allergic dermatitis
13	Painful ears	6 months	Severe diffuse otitis externa	7	Improved
14	Swollen, painful, itching ears	2 days	Severe diffuse otitis externa	4	Improved
15	Itching, crusted, swollen right ear	1 week	Diffuse otitis externa with crusting	1	Not known
16	Crusted, weeping left ear	4 days	Diffuse otitis externa with crusting	2	Improved
17	Itching and discharging ears	6(?) months	Diffuse otitis externa	4	Improved
18	Itching, discharging ears	1 year	Diffuse otitis externa with crusting	1	Not known

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
19	Itching ears	6(?) months	Diffuse otitis externa with discharge	4	Improved
20	Itching, discharging ears	6(?) weeks	Diffuse otitis externa with discharge	2	Improved
21	Swelling and weeping ears	6 + months	Diffuse otitis externa	3	Improved
22	Itching and discharging ears	Unknown	Diffuse otitis externa with excoriation	1	Not known
23	Itching and stuffy ears	1 + weeks	Diffuse otitis externa with crusting	1	Not known
24	Crusting, both ears	2 years	Diffuse otitis externa with crusting	10	Improved
25	Crusted and itching ear	3 + months	Diffuse otitis externa with excoriation	15	Improved but marked allergic reaction to treatment

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
26	Itching and discharging ears	Unknown	Otitis externa with discharge	5	Improved
27	Crusted and itching ears	2+ years	Diffuse otitis externa with crusting	6	Improved
28	Swollen and painful ears	4+ days	Diffuse otitis externa with unlocalized furuncles	4	Improved
29	Itching and discharging ears	3 months	Diffuse otitis externa with crusting	3	Improved
30	Itching and crusting right ear	3+ months	Diffuse otitis externa with crusting	4	Improved (Allergic factos also)
31	Discharging and itching ears	3 months	Diffuse otitis externa with foul debris	2	Improved

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
32	Stuffy and uncomfortable left ear	Many years	Old radical cavity	3	Improved
33	Painful and bleeding right ear	1 day	Traumatic laceration canal and drum	13	Complete healing of perforation
34	Injury left ear by tree limb	1 day	Abrasion canal wall perforation of drum	3	Improved, no infection
35	Discharging right ear	10 years	OMSCH, central perforation	5	Ear dry, apparent closing of perforation
36	Discharging right ear	2 weeks	Otitis media, large central perforation	3	Ear dry
37	Discharging right ear	From childhood	OMSCH, central perforation	3	Ear dry
38	Discharging left ear	6+ months	Large double perforation, left drum	3	Improved (TB Patient)
39	Discharging right ear	9 days	OMSCH, central perforation	7	Ear dry
40	Painful, discharging left ear	15 years	Otitis externa & OMSCH, central perforation	6	Ear dry

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
41	Discharging left ear	Many years	OMSCH, marginal perforation	10	Ear dry
42	Fullness, irritation left ear	15 years	Left radical mastoid cavity	-	Improved
43	Pain, deafness left ear	2 weeks	OMSA, central perforation	6	Improved
44	Discharging left ear	Many years	OMSCH, marginal perforation, granulations	5	Ear dry
45	Discharging right ear	Many years	Radical mastoid cavity	-	Improved
46	Discharging left ear	Many years	Left radical mastoid cavity	-	Unimproved tubal discharge continues
47	Discharging right ear	3 months	OMSCH, central perforation	18	Ear dry
48	Bloody discharge right ear	Many years	OMSCH, marginal perforation	-	Allergic reaction mastoid surgery advised

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
49	Discharge left ear	8 years	OMSCH, marginal perforation with granulations	7	Ear dry
50	Running right ear	6 months	OMSCH, marginal perforation	1 year	Unimproved patient did not return
51	Itching discharging right ear	Many years	Modified radical mastoid cavity	7	Ear dry
52	Irritated discharge left ear	13 years	Radical mastoid cavity	11	Improved but allergic reaction occurred
53	Discharge right ear	10 years	OMSCH, marginal perforation	8	Ear dry
54	Discharge left ear	many years	OMSCH, central perforation	4	Improved
55	Discharge right ear	10 days	OMSCH, central perforation	7	Ear dry
56	Discharging painful left ear	many years	OMSCH, central perforation	5	Improved

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
57	Discharge deafness left ear	many years	OMSCH, marginal perforation	8	Ear dry
58	Discharge deafness both ears	many years	Right OMSCH with cholesteatoma Left OMSCH, marginal perforation	3	Improved
59	Discharge right ear	3 days	OMSCH, central perforation. Left same	4	Ear dry
60	Discharge left ear	1 week	OMSCH, central perforation	4	Improved
61	Deafness right ear	1 week	Large perforation right drum, traumatic following blow	9	Drum healed and intact

in the remarkable protection afforded by the normal human skin against bacterial invasion. These factors, however, are imperfectly understood, and further basic study is necessary.

"A factor of considerable importance is the pH of the skin. The surface of the skin is enveloped by an 'acid mantle,' which exhibits pronounced defense against bacterial invasion. Determinations of the pH of the skin by other investigators reveal the following facts: *1.* The seborrheic areas of the body are more alkaline than other parts of the skin's surfaces; *2.* the alkaline areas are less bactericidal than the rest of the skin, thus being predisposed to cutaneous infection; *3.* in most cases of vesicular eczema there is a shift of the pH of the skin toward the alkaline side; *4.* the highest degree of alkalinity is present in fissures associated with eczematous processes, and *5.* moisture, dead horny tissue and relative alkalinity are four favorite conditions for the propagation of many forms of fungi."

In view of the foregoing considerations, it would seem that the ideal agent for the treatment of aural discharge should have the following properties: *1.* it should be strongly effective against Gram-negative organisms; *2.* the vehicle used with the agent must not impair its efficiency; *3.* the agent should be easy to apply and should not interfere with the elimination of discharge or debris; *4.* it should be in an acid medium, and *5.* it should have a drying effect.

Reports on the use of streptomycin and its modification, dihydrostreptomycin, have occurred with increasing frequency in the medical literature since 1947. Pulaski and Matthews[12] reported favorable results in five cases of chronic otitis externa, treated with a solution of streptomycin (5 micrograms per cubic centimeter). They also treated 13 patients with chronic otitis media and reported that 11 were benefited. They suggested that greater concentrations of the antibiotic agent might prove of further benefit.

Callaway[13] reported dramatic improvement of a case of severe infection of the ear by *Pseudomonas seruginosa* that

had proved resistant to many forms of treatment over a period of nearly one year. He used an aqueous solution of streptomycin containing 2,500 units per cubic centimeter.

Lundon[14] treated a series of 20 outpatients of the Queen Mary Veterans' Hospital in Montreal, Canada, all of whom had chronic suppurative otitis media. The duration of treatment varied from one to 12 weeks. Of this group, 15 responded favorably, "as evidenced by 'dry ears' and absence of pathogenic bacteria"; five cases were regarded as therapeutic failures.

Miller *et al.*[15] used dihydrostreptomycin ointment, 5 micrograms per gram, in the treatment of 124 cases of pyogenic infections of the skin. Nine of these were diagnosed as otitis externa. These authors demonstrated that formulas of oil in water and greaseless base yielded a superior release of the antibiotic. They also emphasized the fact that good results with dihydrostreptomycin usually occurred rapidly, but that prolonged use of the agent was not advocated, since poor results might be due to drug-fastness. The treatment caused a vesicular dermatitis in four patients of the series (3.7 per cent).

Harris *et al.*[16] presented bacteriological and clinical observations made in 27 ears "with suppurative otitis, under treatment with local instillations of streptomycin solutions. Clinical improvement occurred in about half these ears. Drainage stopped during the treatment and did not recur during a follow-up period of from two to six months." They used streptomycin in sterile, isotonic sodium chloride solution, with concentrations varying from 1,000 to 25,000 units per cubic centimeter. They noted that resistance of organisms, previously sensitive to streptomycin, appeared during the course of treatment in a large number of cases, despite the fact that the concentration of streptomycin instilled was at least 20 times greater than the minimum concentration required to inhibit the growth of the organisms *in vitro*, by the usual sensitivity tests, before treatment was started.

Senturia and Broh-Kahn[17] treated a series of 54 consecutive cases of diffuse otitis with streptomycin, incorporated in "Carboprop" [60 parts of Carbowax (4,000) and 40 parts of propylene glycol]. Favorable results were obtained when a concentration of 5 mg. of streptomycin per gram was used. When concentrations of 1 mg. of streptomycin per gram, or less, were used the results were either poor or inconclusive. Controls treated with "Carboprop" alone did not produce a comparable improvement.

CLINICAL AND LABORATORY INVESTIGATION.

To date, all available reports dealing with the topical use of streptomycin have recorded the use of this antibiotic in an aqueous solution or in some form of ointment or similar medium. In May, 1949, following the conclusion of a scientific session at a national meeting, a group of otologists were informally discussing the treatment of various conditions. At that time, someone suggested the use of streptomycin incorporated in boric acid powder. In view of the fact that a drying agent—such as boric acid—has always been one of the most successful in the author's experience, he decided to begin an extended clinical trial of such a method of therapy.

The method of treatment of aural discharge, devised for this clinical study, is as follows: *1.* the canal is carefully cleaned of all debris, using 70 per cent ethyl alcohol; it is then dried; *2.* dihydrostreptomycin-boric acid powder (one part of the former, three parts of the latter) is insufflated generously into the canal, using a DeVilbiss powder blower; *3.* the patient is instructed to leave the ear untouched until the following morning, and at that time he is to start the use of five drops of boric acid-alcohol and is to continue this three times daily until the next office visit. Office visits were spaced at two or three times a week whenever possible, but this schedule does not hold for all the patients included in this report. Throughout the years, boric acid-alcohol drops have remained a standby with many otologists. The formula may vary slight-

ly among otologists, but the prescription given the patients in the present report is as follows: Ethyl alcohol, 70 per cent; boric acid powder, quantity sufficient to saturate.

In view of the relative importance of maintaining the acidity of the skin, the pH of the substances used in this clinical investigation was determined* with the following results:

Two hundred mg. of dihydrostreptomycin-boric acid powder der mixture, dissolved in 5 ml. distilled water (practically all of which when into solution)—pH 4.32.

Boric acid powder, U. S. P.—pH 4.05.

Boric acid-solution—pH 5.50.

Boric acid-alcohol solution, diluted one-half with distilled water— pH 5.50.

These values demonstrate that both of the agents used in this study assist in maintaining the acid condition of normal skin, and it may be that the major value of boric acid-alcohol drops can be attributed to this factor.

BACTERIOLOGICAL STUDIES.

Pure cultures of laboratory strains of *Pseudomonas aeruginosa* and *Staphylococcus aureus* were streaked on agar plates and were then treated with dihydrostreptomycin-boric acid powder and with boric-alcohol solution. In the former culture plates, wide areas of bacterial inhibition were clearly visible, but in the latter instance, bacteriostatic action was extremely limited. Fig. 1 demonstrated the bacteriological results of typical laboratory studies made with the agents described and employed in this clinical study.

The observed *in vitro* action of dihydrostreptomycin alone was in accord with reports of other investigators, but nothing in the literature has suggested the enhanced bacteriostatic action of the dihydrostreptomycin-boric acid powder mixture.

*All pH determinations were made using the Beckman Glass Electrode pH Meter (Model G), with solutions at a room temperature of 23° Centigrade.

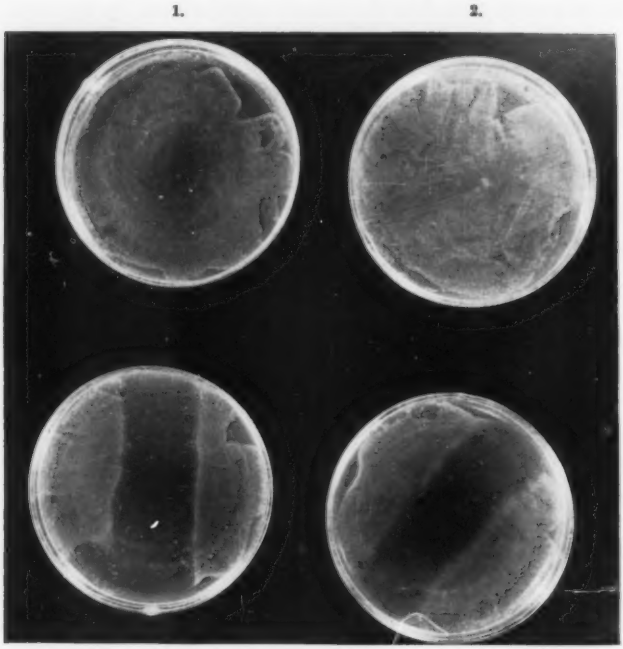

Fig. 1. Pus collected on a swab from a patient with chronic mastoiditis was suspended in 1 ml. of infusion broth. One drop of the suspension was placed in the center of each of four extract-agar and four blood-agar plates and spread over the surface of the plates with a glass rod. Ordinary streak plates of the suspension were made at the same time on extract agar and blood agar.

One extract and one blood-agar plate were left as controls. A loopful of powdered boric acid was streaked across the center of one extract and one blood-agar plate; a loopful of powdered dihydrostreptomycin was streaked across another similar set; and dihydrostreptomycin-boric acid powder mixture was streaked across a third set. All of the plates were incubated for 24 hours.

The illustration shows the appearance of the extract-agar plates after incubation.

Plate 1. Control.

Plate 2. With powdered boric acid.

Plate 3. With powdered dihydrostreptomycin.

Plate 4. With dihydrostreptomycin-boric acid powder mixture.

The streak plates contained colonies of the following species: Pseudomonas aeruginosa, Aerobacter aerogenes, hemolytic Staphylococcus aureus, Streptococcus fecalis, and a diphtheroid which was not completely identified. The colonies appearing in the area of inhibition on Plate 3 were the diphtheroid and Streptococcus fecalis.

It has been pointed out that streptomycin is more effective when used in alkaline medium.[18] While this may be true when used in dilute solutions, the present studies suggest that it is also strongly effective in high concentrations, even though incorporated in markedly acid media.

CLINICAL REPORTS.

Case histories taken from a private practice seldom are ideal or satisfactory for statistical analysis. Controls usually cannot be used, and the factors of time, economical situation of the patients, and the difficulty of obtaining follow-up data in many instances often result in incomplete analyses. Patients who receive substantial benefit from the therapy used frequently fail to return for a check-up when their major complaints are relieved. This results in incomplete reports of cure and recurrence of symptoms.

Since bacteriological data in these presently reported cases were in general well established, bacteriological cultures were not made routinely, but in those cases in which this was done, the findings were in accord with those given in the literature as herein reviewed.

The cases observed present a variety of conditions that do not lend themselves to a tabular chart report; therefore, each case has been abstracted as briefly as possible, and all findings not directly related to the chief complaint have been omitted. For the purpose of simplification, the dihydrostreptomycin-boric acid powder, which was used for treatment in this series of patients, has been named "strepto B," and the boric acid-alcohol drops have been designated as "B and A drops." These terms are used throughout the case reports.

Otitis Externa.—It is generally accepted that factors other than bacterial infection may play a part in many cases of otitis externa. Allergy and other constitutional disorders must not be overlooked as etiological agents. When, however, the discharge is reduced or stopped after two or three treatments with an antibacterial agent, it seems reasonable to assume that in these cases infection is the major factor.

It has been found difficult to fit the following cases into any formal classification and, therefore, several descriptive terms have been used in specific instances instead. Some authors have used such terms as "suppurative" and "eczematoid" for categorical, differentiation purposes, but these terms are not generally understood or accepted, and so are not used here.

SUMMARY AND CONCLUSIONS.

1. Dihydrostreptomycin-boric acid powder appears to be an effective agent in the treatment of aural discharge.

2. The acidity of this preparation has been determined. Evidence taken from the literature shows that acidity is desirable in the treatment of aural discharge.

3. Bacteriological evidence from the present study suggests that this mixture is more effective than is dihydrostreptomycin alone.

4. Moisture, which encourages bacterial growth, is reduced by the drying effect of this treatment.

5. Vesicular skin lesions occurred in a small percentage of cases when this treatment was used, but they cleared rapidly upon cessation of the application.

6. Thirty-one cases of otitis externa were treated with dihydrostreptomycin-boric acid powder. In 21 of these cases improvement was obtained. In seven cases results were unsatisfactory or inconclusive. In three cases vesicular dermatitis occurred.

7. Thirty cases of otitis media, mastoiditis or tympanic trauma were treated with dihydrostreptomycin-boric acid powder. In 22 of these cases improvement was noted. In six cases results were unsatisfactory or inconclusive. In two cases vesicular dermatitis occurred.

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BOOK REVIEW.

Atlas der Hals- Nasen- Ohren-Krankheiten. By C. v. Eicken and A. Schulz van Treeck. Third Edition. 199 pages with 470 illustrations, many of them in color. Stuttgart: Georg Thieme, Verlag. 1951. Price DM 78. American agents, New York: Grune & Stratton, Inc.

The reproduction of colored drawings and photographs in this atlas is beautiful, although in many instances the coloring is too vivid. The detailed work in most of these drawings is highly commendable.

There are many conditions shown which, in our present day therapy, in this country, are not seen in the advanced stages illustrated. One has the feeling, after turning through the pages, that the atlas is not as up to date as it should be. Many of the descriptions accompanying the illustrations are too brief, and yet ample space is present on many pages.

Such an atlas should be of value to any student beginning a study of diseases of the ear, nose and throat. W. P. C.

THE GRADUATE SCHOOL OF MEDICINE OF THE UNIVERSITY OF FLORIDA.

The sixth annual University of Florida Midwinter Seminar in Ophthalmology and Otolaryngology will be held Jan. 14-19, 1952. As in previous years, Miami Beach will be the host city.

The lectures on ophthalmology will be presented on Jan. 14, 15 and 16; those on otolaryngology on Jan. 17, 18 and 19. Lecturers for the courses in ophthalmology will include such distinguished teachers as Dr. Alson E. Braley, Iowa City, Iowa; Dr. John M. McLean, New York City; Dr. Wendell Hughes, Hempstead, Long Island, New York; Dr. Irving H. Leopold, Philadelphia, Pa., and Dr. Bruce Fralcik, Ann Arbor, Mich.

Outstanding otolaryngologists who will lecture include Dr. Fred W. Dixon, Cleveland, Ohio; Dr. Thomas D. Galloway,

Evanston, Ill.; Dr. Anderson G. Hilding, Duluth, Minn.; Dr. Moses H. Lurie, Boston, Mass., and Dr. Henry L. Williams, Rochester, Minn.

The Southern Section Meeting of the American Laryngological, Rhinological and Otological Society will be held Monday, Jan. 14, at Atlanta, Ga. The Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology planned its meeting in Havana the week following our seminar. Registrants of the seminar might like to include one or both these other meetings in their southern trip.

All meetings will be held at the Sans Souci Hotel. Registration fee for the seminar is $40.00. A check for $10.00, payable to the University of Florida Midwinter Seminar, must accompany your application. This is not returnable. The remainder of the registration fee will be paid at the seminar desk at the Sans Souci Hotel on your arrival.

Convention rates are good for the seminar and for any period before Jan. 14, but after Jan. 19 the regular, higher rates are effective.

Division of Ophthalmology: Shaler Richardson, M.D., Jacksonville; Nelson M. Black, M.D., Miami; Charles Boyd, M.D., Jacksonville; Bascom Palmer, M.D., Miami.

Division of Otolaryngology: Marshall Taylor, M.D., Jacksonville; Walter T. Hotchkiss, M.D., Miami Beach.

MISSISSIPPI VALLEY MEDICAL SOCIETY 1952 ESSAY CONTEST.

The Twelfth Annual Essay Contest of the Mississippi Valley Medical Society will be held in 1952. The Society will

offer a cash prize of $200.00, a gold medal, and a certificate of award for the best unpublished essay on any subject of general medical interest (including medical economics and education) and practical value to the general practitioner of medicine. Certificates of merit may also be granted to the physicians whose essays are rated second and third best. Contestants must be members of the American Medical Association and residents and citizens of the United States. The winner will be invited to present his contribution before the Seventeenth Annual Meeting of the Mississippi Valley Medical Society to be held in St. Louis, Mo., Oct. 1, 2, 3, 1952, the Society reserving the exclusive right to publish the essay first in its official publication — the *Mississippi Valley Medical Journal* (incorporating the *Radiologic Review*). All contributions must be typewritten in English in manuscript form, submitted in five copies, not to exceed 5,000 words, and must be received not later than May 1, 1952. The winning essays in the 1950 contest appear in the January, 1951, issue of the *Mississippi Valley Medical Journal* (Quincy, Ill.). Further details may be secured from Harold Swanberg, M.D., Secretary, Mississippi Valley Medical Society, 209-224 W. C. U. Building, Quincy, Ill.

THE FOUNDATION OF THE AMERICAN SOCIETY OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

The Foundation of the American Society of Plastic and Reconstructive Surgery announced the winners of this year's awards in its Competitive Essay, at the annual meeting of the Society in Colorado Springs, Oct. 30-Nov. 1, 1951.

Dr. Richard B. Stark, New York City, won a Scholarship Award for his essay: "The Cross-Leg Flap Procedure."

Dr. Kathryn Lyle Stephenson, Santa Barbara, Calif. Essay: "The Production of Ectopic Cartilage." Honorable Mention.

Dr. Michael N. Tempest, Leeds, Yorkshire, England. Essay: "Cross-Finger Flaps in the Treatment of Injuries to the Finger Tip." Honorable Mention.

Dr. Allyn J. McDowell, North Hollywood, Calif. Essay: "Mass Treatment of Burns in Atomic Warfare." Honorable Mention.

The Award Committee consisted of: Dr. Robert H. Ivy (Philadelphia), Dr. Neal Owens (New Orleans), Dr. Kenneth L. Pickrell (Durham, N. C.), Dr. Lyndon A. Peer (Newark), Dr. Clarence R. Straatsma (New York) and Dr. Jacques W. Maliniac, Ex-Officio (New York).

For full particulars pertaining to the 1952 Competitive Essay write to the Award Committee, care of Jacques W. Maliniac, M.D., 11 East 68th Street, New York 21, N. Y.

COURS INTERNATIONAL D'AUDIOLOGIE CLINIQUE.

An International Course in Clinical Audiology and the Measurement and Correction of Deafness will be given in the Grand Amphitheatre de la Faculte de Medecine, Paris, Feb. 11-17, 1952.

Course will include Physiology, Physics and Psycho-Physics of Hearing; Audiometric and Speech Tests, Technique and Interpretation; Classification and Studies of Deafness; Surgical Treatments and Prothesis.

For further information address the Secretary, Dr. J. E. Fournier, 164 Boulevard Haussmann, Paris (VIII°), France.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

December 1, 1951.

Audicon Models 400 and 415.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Audivox Model Super 67.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dahlberg Model D-1.
Manufacturer: The Dahlberg Co., 2730 W. Lake St., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Star Model.

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model 45.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Model J (Tiny-Myte); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Rochester Model R-1; Rochester Model R-2.

Manufacturer: Rochester Acoustical Laboratories, Inc., 117 Fourth St., S.W., Rochester, Minn.

Silvertone Model 103BM.
Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.
Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model J-92.
Manufacturer: Sears-Roebuck Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model P-15.
Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.
Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.
Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.
Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 1700.
Manufacturer: Telex, Inc., Telex Park, Minneapolis 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube Model No. 300.
Manufacturer: Trimm, Inc., 400 Lake St. (P. O. Box 489), Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., successor to Western Electric Hearing Aid Division, 259 W. 14th St., New York 11, N. Y.

Zenith Model 75; Zenith Miniature 75; Zenith Model Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse, N. Y.
Vice-President: Albert C. Furstenberg, University Hospital, Ann Arbor, Mich.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Meeting: Royal York Hotel, Toronto, Canada, May 18-19, 1952.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Royal York Hotel, Toronto, Canada, May 23-24, 1952.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President Dr. C. Stewart Nash, 108 Medical Arts Bldg., Rochester, N. Y.
Meeting: Royal York Hotel, Toronto, Canada (mornings only), May 20-22, 1952.

MID-WINTER MEETINGS.

Eastern Section: Waldorf-Astoria, New York City, Jan. 11, 1952.
Council Meeting: Waldorf-Astoria, New York City, Jan. 12, 1952.
Southern Section: Academy of Medicine, Atlanta, Ga., Jan. 14, 1952.
Middle Section: Indianapolis Athletic Club, Indianapolis, Ind., Jan. 28, 1952.
Western Section: County Medical Bldg., Los Angeles, Calif., Jan. 19,20, 1952.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

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AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Royal York Hotel, Toronto, Canada, May 13-16, 1952.

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Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

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OTOSCLEROSIS STUDY GROUP.

Meeting: Palmer House, Chicago, Ill., October, 1952.

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Pres.: Dr. José Gros.
Sect.: Dr. Pedro Hernandez Gonzalo, Calle 8, 358, Havana, Cuba.
Meeting: Third Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Havana, Cuba, January 20-24, 1952.

THIRD LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

Time and Place: Caracas, Venezuela, 1953.

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Meeting: St. Louis, Hotel Jefferson, Oct. 1-3, 1952.

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Secretary-Treasurer: Dr. Victor Goodhill.
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Secretary of Section on Ophthalmology: Dr. Carroll McCoy.
Chairman of Section on Otolaryngology: Dr. Howard P. House.
Secretary of Section on Otolaryngology: Dr. Edwin Scobee.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

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